ACUTE FEBRILE ENCEPHALOPATHY- CLINICAL PROFILE AND OUTCOME

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I hereby declare that the thesis entitled "**ACUTE FEBRILE ENCEPHALOPATHY- CLINICAL PROFILE AND OUTCOME**" submitted towards partial fulfillment of the requirements for the award of **M.D. in General Medicine** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Rajesh Mahajan,** Professor, Department of Medicine, **Dr. Veenu Gupta**, Professor & Head, Department of Microbiology, & **Dr. Rajinder K Bansal,** Professor, Department of Neurology. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

to

my Family

&

Teachers

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COMMIT TO THE LORD WHATEVER YOU DO, AND HE WILL ESTABLISH YOUR PLANS. Successful completion of a task is often less due to individual talent than to the fact that the person has been blessed by the 'GOD ALMIGHTY'

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Dr. Mehak Arora

ABBREVIATIONS

AFE	Acute Febrile Encephalopathy
SAE	Sepsis Associated Encephalopathy
TBM	Tubercular Meningitis
DAMA	Discharge Against Medical Advise
GPE	General Physical Examination
PRES	Posterior Reversible Encephalopathy Syndrome
CBNAAT	Cartridge Based Nucleic Acid Amplification Test
CSF	Cerebrospinal Fluid
ADA	Adenosine deaminase
HSV	Herpes Simplex Virus
PCR	Polymerase Chain Reaction
C/S	Culture and Sensitivity
CAD	Coronary Artery Disease
CHF	Congestive Heart Failure
COAD	Chronic Obstructive Airway Disease
EEG	Electroencephalogram
PCT	Procalcitonin
UTI	Urinary Tract Infection
RTI	Respiratory Tract Infection
SSTI	Skin and Soft Tissue Infection
TLC	Total Leucocyte Count
EVD	External Ventricular Drain
ICH	Intra Cerebral Hemorrhage
LFT	Liver Function Test
RFT	Renal Function Test
VAP	Ventilator Associated Pneumonia

ABSTRACT

INTRODUCTION: Acute febrile encephalopathy is a clinical spectrum where a short febrile illness is accompanied or followed by altered mental state and is characterized by diffuse disease of brain that alters it's structure or function manifested by a combination of drowsiness, disorientation, seizures or coma.

AIM OF STUDY: To study etiological profile of patients presenting with fever accompanied or followed by altered mental status and to study outcome of such patients.

MATERIALS AND METHODS: This was an observational prospective study conducted on all the patients who presented with complaints of fever accompanied or followed by altered sensorium fitting into the inclusion criteria of age more than 18 years and excluding CVA, metabolic causes independently explaining the presentation were enrolled in the study in the Department of Medical Emergency of DMCH, Ludhiana over a period of 1 and half years. Outcome of the patients was assessed at the time of discharge.

RESULTS: In our study it was found that SAE was the most common etiology of acute febrile encephalopathy (36.5%) followed by TBM (29.2%) and Viral meningitis/ meningio-encephalitis (12.2%).Dengue associated Encephalopathy/ Encephalitis was found in 11.1% patients. Pyogenic Meningitis was found amongst 4.4% cases. Other rare etiologies like Scrub, Leptospira associated neurological complications, Post covid Encephalopathy and Cryptococcal Meningitis were also rarely found. The maximum recovery rate was found in patients with TBM (35.9%) Also, out of the total deaths observed, maximum mortality rate was found in patients with Sepsis associated Encephalopathy (43.2%).

CONCLUSION: Acute Febrile Encephalopathy is a broad term inclusive of many variable etiologies. Early diagnosis and prompt treatment in these patients can impact the survival of these patients especially in the setting of a tertiary hospital care.

KEYWORDS: Meningitis, Encephalitis, TBM, Sepsis Associated Encephalopathy, CSF, MRI

TABLE OF CONTENTS

S. NO.	TOPIC	PAGES
I	INTRODUCTION	1-6
II	AIMS AND OBJECTIVES	7
III	REVIEW OF LITERATURE	8-34
IV	MATERIALS AND METHODS	35-37
V	OBSERVATIONS AND RESULTS	38-96
VI	DISCUSSION	97-105
VII	SUMMARY AND CONCLUSIONS	106-108
VIII	BIBLIOGRAPHY	109-121
	APPENDICES – I	
	- PROFORMA	
	- MASTER CHART	
	APPENDICES – II	
	- APPROVAL LETTER OF THESIS PLAN	
	- THESIS PLAN	

LIST OF TABLES

Table No	Title	Page No.
1	VARIOUS STUDIES CONDUCTED FOR ACUTE PYOGENIC MENINGITIS IN ADULTS	21
2	AGE DISTRIBUTION OF PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY	38
3	SEX DISTRIBUTION OF PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY	39
4	PRESENTING COMPLAINTS OF PATIENTS WITH AFE	40
5	CO-MORBIDITIES IN STUDY GROUP PATIENTS (n-271)	41
6	ETIOLOGICAL PROFILE OF PATIENTS WITH AFE	42
7	CT HEAD FINDINGS IN PATIENTS OF AFE ENROLLED IN THE STUDY	44
8	TABLE SHOWING MRI FINDINGS OF THE PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY	45
9	AGE DISTRIBUTION IN TBM PATIENTS	49
10	SEX DISTRIBUTION IN TBM PATIENTS	49
11	CHIEF COMPLAINTS OF PATIENTS WITH TBM	50
12	TABLE SHOWING CBNAAT IN CSF POSITIVE CASES AMONGST TOTAL CASES OF TUBERCULOUS MENINGITIS.	51
13	CSF ADA LEVELS IN PATIENTS WITH TBM	52
14	CSF PROTEIN LEVELS IN PATIENTS WITH TBM	53
15	CSF: SERUM GLUCOSE <0.5IN TBM PATIENTS	54
16	SENSITIVITY OF CSF ADA IN DIAGNOSING TBM	55
17	SENSITIVITY OF CSF GLUCOSE IN DIAGNOSING TBM	55
18	SENSITIVITY OF CSF PROTEIN IN DIAGNOSING TBM	56
19	AGE DISTRIBUTION IN PYOGENIC MENINGITIS	57
20	SEX DISTRIBUTION IN PYOGENIC MENINGITIS	57
21	CSF FINDINGS IN PYOGENIC MENINGITIS	58
22	MRI FINDINGS IN PYOGENIC MENINGITIS	59
23	AGE DISTRIBUTION IN VIRAL MENINGO-ENCEPHALITIS	60

24	SEX DISTRIBUTION IN VIRAL MENINGO-ENCEPHALITIS	60
25	PRESENTING COMPLAINTS IN VIRAL MENINGO-ENCEPHALITIS	61
26	HSV PCR POSITIVE CASES AMONGST TOTAL VIRAL MENINGO-ENCEPHALITIS PATIENTS	61
27	VIRAL MENINGO-ENCEPHALITIS PATIENTS WITH CSF HSV PCR POSITIVE (TOTAL = 16)	62
28	VIRAL MENINGITIS/ENCEPHALITIS PATIENTS WITH NEGATIVE HSV PCR (TOTAL = 17)	62
29	AGE DISTRIBUTION OF PATIENTS WITH SAE	63
30	SEX DISTRIBUTION OF PATIENTS WITH SAE	64
31	CO-MORBIDITIES IN PATIENTS WITH SAE	66
32	POSITIVE C/S OUT OF TOTAL STUDY POPULATION	67
33	C/S POSITIVITY IN SAE PATIENTS	68
34	TOTAL NUMBER OF INFECTION SITES IN PATIENTS WITH POSITIVE C/S IN SAE	69
35	SOURCES OF ISOLATES OBTAINED IN C/S OF VARIOUS SAMPLES IN PATIENTS WITH SAE	70
36	DISCRIPTION OF ALL THE ISOLATES OBTAINED IN PATIENTS WITH SAE.	71
37	SOURCE OF SEPSIS IN SAE PATIENTS	72
38	SIGNIFICANCE OF PCT IN SAE (n-86)	73
39	HYPOTENSION IN PATIENTS WITH SAE	74
40	HYPOTENSION IN PATIENTS WITH CULTURE POSITIVE SAE	75
41	AGE DISTRIBUTION OF PATIENTS WITH DENGUE ASSOCIATED NEUROLOGICAL COMPLICATIONS	75
42	SEX DISTRIBUTION OF PATIENTS WITH DENGUE ASSOCIATED NEUROLOGICAL COMPLICATIONS	75
43	RAISED HEPATIC TRANSAMINASES IN PATIENTS WITH DENGUE ENCEPHLOPATHY	76
44	DENGUE SHOCK SYNDROME AMONG PATIENTS WITH DENGUE ENCEPHALOPATHY	77

45	AGE DISTRIBUTION OF PATIENTS WITH SCRUB ASSOCIATED ENCEPHALOPATHY	78
46	SEX DISTRIBUTION OF PATIENTS WITH SCRUB ASSOCIATED ENCEPHALOPATHY	78
47	MRI FINDINGS OF PATIENTS WITH SCRUB ASSOCIATED ENCEPHALOPATHY.	78
48	OUTCOME OF EACH ETIOLOGICAL ENTITY AT THE TIME OF DISCHARGE (DIVIDED INTO RECOVERY/DAMA/DEATH)	81
49	STATISTICAL INSIGNIFICANCE OF AGE AS A VARIABLE EFFECTING OUTCOME IN AFE	83
50	STATISTICAL INSIGNIFICANCE SHOWN WHEN GENDER WAS COMPARED IN TERMS OF OUTCOME	84
51	HYDROCEPHALUS ON RADIO IMAGING LEADING TO WORSE OUTCOME IN PATIENTS WITH TBM	85
52	LEPTOMENINGITIS ON MRI NOT AFFECTING OUTCOMEIN TBM	86
53	VASCULITIC INFARCT ON MRI NOT AFFECTING OUTCOME IN TBM	86
54	LOW GLUCOSE IN CSF EFFECTING OUTCOME IN TBM	86
55	CSF FLUID GROWTH HAS NO IMPACT ON OUTCOME	88
56	STATISTICAL INSIGNIFICANCE WHEN HSV RNA PCR POSITIVITY IN CSF WAS COMPARED WITH THE OUTCOME.	89
57	NO AFFECT OF ENCEPHALITIS ON MRI ON OUTCOME OF PATIENTS WITH VIRAL ENCEPHALITIS	90
58	HYPOTENSION LEADING TO ADVERSE OUTCOME IN PATIENTS WITH VIRAL MENINGIO-ENCEPHALITIS.	90
59	NO EFFECT OF TRANSAMINITIS ON OUTCOME OBSERVED.	91
60	HYPOTENSION EFFECTING OUTCOME IN DENGUE ENCEPHALOPATHY PATIENTS	92
61	OUTCOME OF PATIENTS OF SAE WITH TYPE 2 DM	93

62	IMPACT OF TYPE 2 DM ON OUTCOME OF SAE	94
63	SIGNIFICANT IMPACT OF SEPTIC SHOCK ON OUTCOME OF SAE	94
64	EFFECT OF ANEMIA ON OUTCOME IN SAE	95
65	EFFECT OF RENAL/HEPATIC DERANGEMENT ON OUTCOME IN PATIENTS WITH SAE	95

LIST OF FIGURES

Figure. No	Title	Page No.
1	AGE WISE DISTRIBUTION OF PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY	38
2	SEX DISTRIBUTION OF PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY	39
3	OTHER PRESNTING COMPLAINTS IN AFE	40
4	CO-MORBIDITIES IN PATIENTS WITH AFE	42
5	ETIOLOGICAL PROFILE OF PATIENTS HAVING AFE ENROLLED IN THE STUDY	43
6	LEPTOMENINGITIS ON MRI BRAIN DIVIDED INTO ETIOLOGICAL ENTITIES	46
7	VASCULITIC INFARCTS ON MRI BRAIN DIVIDED INTO ETIOLOGICAL ENTITIES	46
8	ENCEPHALITIS ON MRI BRAIN DIVIDED INTO ETIOLOGICAL ENTITIES	47
9	HYDROCEPHALUS ON MRI BRAIN DIVIDED INTO ETIOLOGICAL ENTITIES	48
10	POSITIVE CBNAAT IN CSF CASES OUT OF TOTAL TBM PATIENTS.	51
11	CSF ADA LEVELS IN PATIENTS WITH TBM	52
12	CSF PROTEIN LEVELS IN PATIENTS WITH TBM	53
13	CSF: SERUM GLUCOSE	54
14	MRI FINDINGS IN PATIENTS WITH CBNAAT POSITIVE TBM WITH LOW GCS(<10)	56
15	CSF FINDINGS IN PYOGENIC MENINGITIS	58
16	IMAGING FINDINGS IN PATIENTS WITH PYOGENIC MENINGITIS.	59
17	AGE DISTRIBUTION IN PATIENTS WITH SAE	64
18	SEX DISTRIBUTION OF PATIENTS WITH SAE	65
19	POSITIVE C/S OUT OF TOTAL STUDY POPULATION	67

20	C/S POSITIVITY IN SAE PATIENTS	68
21	SOURCES OF ISOLATES OBTAINED IN C/S OF VARIOUS SAMPLES IN PATIENTS WITH SAE	70
22	SOURCES OF SEPSIS IN PATIENTS WITH SAE BASED ON C/S POSITIVITY AND CLINICAL PRESENTATION.	72
23	SIGNIFICANCE OF PCT IN SAE	73
24	MRI FINDINGS OF PATIENTS WITH SAE67	74
25	RAISED HEPATIC TRANSAMINASES IN PATIENTS WITH DENGUE ENCEPHLOPATHY	76
26	IMAGING IN DENGUE ENCEPHALOPATHY	77
27	OUTCOME OF VARIOUS ETIOLOGICAL ENTITIES IN TERMS OF RECOVERY/DAMA/DEATH AT THE TIME OF DISCHARGE (total no. of cases of each etiological entity as denominator)	82
28	DIFFERENT AGE GROUPS COMPARED WITH EACH OTHER IN TERMS OF OUTCOME	83
29	EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH TUBERCULOUS MENINGITIS.	84
30	HYDROCEPHALUS ON MRI BRAIN LEADS TO ADVERSE OUTCOME	85
31	LOW GLUCOSE EFFECTING OUTCOME IN TBM	87
32	EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH PYOGENIC MENINGITIS	88
33	EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH VIRAL MENINGITIS/ MENINGO-ENCEPHALITIS.	89
34	HYPOTENSION LEADING TO ADVERSE OUTCOME IN PATIENTS WITH VIRAL MENINGIO-ENCEPHALITIS	90
35	EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH DENGUE ENCEPHALITIS/ENCEPHALOPATHY	91
36	HYPOTENSION LEADING TO ADVERSE OUTCOME IN DENGUE ENCEPHALOPATHY	92

37	EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH SEPSISASSOCIATED ENCEPHALOPATHY	93
38	SIGNIFICANT IMPACT OF SEPTIC SHOCK ON OUTCOME OF SAE	94
39	EFFECT OF RENAL/HEPATIC DERANGEMENT ON OUTCOME IN PATIENTS WITH SAE	95

INTRODUCTION

Acute febrile encephalopathy is a clinical spectrum where a short febrile illness is accompanied or followed by altered mental state and is characterized by diffuse disease of brain that alters its structure or function manifested by a combination of drowsiness, disorientation, seizures or coma. [1] Considering the fact that confusion is a key sign of encephalopathy, this symptom accounts for around 2% of the patients in emergency departments.[2]

In febrile illness, CNS infections are a common cause of such presentations in hospitals, etiological agent may be a bacterium, virus or a parasite. Meningitis is an inflammation that affects the three protective membrane layers that cover the brain and spinal cord, called the meninges. The outer layer of the meninges is called the dura mater, followed by the arachnoid mater and the pia mater. The two inner layers (arachnoid and pia mater) are also called the leptomeninges and are separated by the subarachnoid space, which contains cerebrospinal fluid (CSF). Bacteria, viruses, fungi, and non-infectious agents such as drugs can also induce meningitis. Pathogens can reach the CSF through hematogenous spread by two main mechanisms: 1) by infecting immune cells, which in turn carry the pathogen to the nervous system, and 2) by crossing blood capillaries and entering the CSF as free pathogens.[3] Significant seasonal and geographical variations have been found to be closely related to the etiological profile of the disease. Fever with altered mental state may also result from tuberculous meningitis, cerebral malaria (CM), and typhoid encephalopathy, In tropical countries like India, tropical fevers owing to infections like Dengue, Leptospira, Scrub typhus, malaria may also contribute to the burden of encephalopathy and may show a seasonal spike in their presentation.

In a similar background of febrile illness, encephalopathy can be precipitated by systemic infections or sepsis without cerebral inflammation i.e.in the setting of Sepsis Associated Encephalopathy where endogenous inflammation of the brain(encephalitis) is not the

cause of fever and altered sensorium. Sepsis associated encephalopathy (SAE) is defined as a diffuse brain dysfunction secondary to infection elsewhere in the body without overt CNS infection. Moreover, sepsis can lead to altered sensorium secondary to systemic complications like hypoglycemia, hypovolemia, hepatic or renal failure, hence these possibilities are to be kept as differentials before attempting to treat these patients.[1]

Patients with meningitis usually suffer from fever, chills, abdominal pain, nausea, and headache [4]. Other manifestations of the disease include short and fast breathing, loss of appetite, neck stiffness and pain, and sensitivity to bright light [5]. In addition, difficulty in focusing or concentrating as well as double vision can also be associated with meningitis and may continue after recovery [6]. Skin rash is also observed with certain types of meningitis and is considered a late sign of bacterial meningitis, where the pathogen in the blood targets the capillary cells, leading to their damage.[7]

In 2013, the International Encephalitis Consortium (IEC) suggested consensus criteria for case definition of encephalitis or encephalopathy of presumed infectious or autoimmune etiology.[8] Neurologic dysfunction in the form of altered mental status lasting for over 24 hours without an alternative cause must be present. Minor criteria that must be present (two for possible and at least three for probable or confirmed) include “fever ≥38°C within 72 hours, seizures, new focal neurologic findings, CSF pleocytosis (≥5 white blood cells [WBCs/μL), neuroimaging with brain parenchymal changes or electroencephalogram (EEG) consistent with encephalitis.

ETIOLOGY OF ACUTE ENCEPHALTIS SYNDROME [9]

I. Viral agents that are known to cause encephalitis

a. <u>Arboviruses, togaviruses, and alphaviruses</u>

- Western equine encephalitis virus
- Eastern equine encephalitis virus
- Venezuelan equine encephalitis virus

b. Flaviviruses (mosquito-borne)

- Japanese encephalitis virus
- St Louis encephalitis virus
- West Nile virus
- Murray Valley encephalitis virus
- Non-arthropod-borne togavirus – rubella virus, Bunyaviruses
- California encephalitis virus Reoviruses
- Colorado tick fever encephalitis virus

c. Herpesviruses

- Herpes simplex 1 and 2
- Varicella zoster virus
- Epstein–Barr virus
- Cytomegalovirus
- Human herpesvirus – 6

d. Enteroviruses

- Polioviruses
- Coxsackie viruses
- Echoviruses
- Enteroviruses 70 and 71

e. Orthomyxoviruses

- Influenza viruses

f. Paramyxoviruses

- Measles virus
- Mumps virus
- Parainfluenza viruses
- Nipah virus

g. Adenoviruses

h. Parvoviruses

i. Rhabdoviruses

II. Non-viral agents

a. Rickettsia:

- Rocky Mountain spotted fever
- Endemic and epidemic typhus
- *Coxiella burnetti*
- Ehrlichiosis
- Scrub typhus

b. Bacterial:

- Pyogenic (bacterial) meningitis
- Tuberculous meningitis
- *Mycoplasma pneumoniae*
- *Listeria monocytogenes*
- Spirochetes: syphilis, Leptospirosis,
- Lyme disease
- Brucellosis
- Legionella
- *Salmonella typhi*
- Cat scratch disease (Bartonellosis)

c. Fungi:

- Cryptococcus
- Histoplasma
- Aspergillus
- Mucormycosis
- Candida
- Coccidiomycosis

d. Protozoa:

- Naeglaria
- Acanthamoeba
- Toxoplasma gondii

e. Metazoa:

- Trichinosis
- Echinococcus
- Cysticercus
- Schistosoma

III. Non-infectious inflammation of brain

- Acute disseminated encephalomyelitis
- Antibody-associated encephalitis
- Collagen vascular disorders

IV Infectious encephalopathy

- Cerebral malaria
- Shigella encephalopathy
- Dengue encephalopathy
- Sepsis syndrome
- Enteric encephalopathy

It is important for the emergency clinicians to accurately diagnose and administer antimicrobials and adjunctive therapies to those who need them while attempting to wait for results of invasive testing. Hence it is important to determine the etiologic spectrum of febrile encephalopathy syndrome, with an emphasis on the CNS infection by focusing on epidemiology and age groups. The knowledge of these data is essential for protocol development at the regional level in order to appropriately manage patients. Irrational use of antibiotics should be avoided in patients who will not be benefitted by any such therapy (alternate etiology). Focal neurological deficits go in favor of Encephalitis (inflammation induced brain damage) as compared to other causes of encephalopathy but further investigations are required to confirm the diagnosis. Early decision making and rapid institution of therapy can be life-saving. In untreated cases, mortality is very high and the survivors are often left with disabling neurologic sequelae. Many a times even a detailed diagnostic workup may not identify a specific organism.

Nevertheless, a detailed examination and workup is warranted as many conditions, such as herpes simplex encephalitis (HSE) and cerebral malaria are eminently treatable. CT scan of the brain should be performed as a baseline investigation, to r/o contraindications of CSF and it may also be s/o meningeal enhancement (on contrast enhanced CT). Since meningeal enhancement is not a specific sign of meningitis and Acute Meningitis may present without showing abnormalities on CT, further investigations like CSF, MRI Brain to be considered afterwards. MRI in cases of HSV and JE may have characteristic findings. A GCS and/or a focal neurological deficit at hospital admission have been found to be independent clinical markers of bad outcomes among children with AFE. Raised intracranial pressure may be contributing to mortality in patients with meningoencephalitis, hence early diagnosis can be helpful. Most acutely ill febrile patients with encephalopathy can make complete neurological recovery once the underlying cause is identified and treated promptly and appropriately, but considerable skill and knowledge is required to distinguish the various groups. [1] This study has been carried out to evaluate the patients presenting with AFE in a tertiary care center in Northern India to understand the etiology, prevalence, any seasonal variation, and their outcomes and changes in etiology and outcome related trends as compared to the previous studies. This study is to be conducted over a period of 1 and half year.

AIMS & OBJECTIVES

- To study etiological profile of patients presenting with fever accompanied or followed by altered mental status.
- To study outcome of such patients with different etiologies for their presenting complaints.

REVIEW OF LITERATURE

Acute febrile encephalopathy is a clinical scenario where patient presents with history of a short febrile illness accompanied or followed by altered mental state. Encephalopathy is characterized by diffuse disease of brain that alters it's structure or function[1], list of differentials in such patients with fever and altered mental state is potentially long ranging from pyogenic meningitis, viral encephalitis, tuberculous meningeal involvement to cerebral malaria, dengue viral encephalopathy, Leptospira related aseptic meningoencephalitis and aseptic meningitis, SAE (Sepsis Associated Encephalopathy, HIV related neuronal complications and other causes. Symptoms and signs of meningitis including one or more of the following: headache, irritability, vomiting, fever, neck stiffness, convulsions, focal neurological deficits, altered consciousness, or lethargy.

Below is a brief overview of possible causes of Acute Febrile Encephalopathy.

TUBERCULOUS MENINGITIS

Tuberculous meningitis is the most severe form of tuberculosis and causes substantial morbidity and mortality in adults and children. The outcome of this disease is especially grave in patients with HIV.[10] TBM represents roughly 1% of all cases of TB and approximately 5% of extra-pulmonary TB cases, but is disproportionately important because it kills or severely disables about half of the people affected.[11]

CLINICAL FEATURES:

TBM typically presents as a sub-acute or chronic meningitis, with many days or weeks of headache, fever and vomiting, culminating in loss of consciousness, focal neurological deficit and death. Occasionally, TBM can present acutely, with these normally late signs already apparent and without a distinct prodromal period. Several studies have defined the clinical features most predictive of TBM. The strongest of these features, across all studies, is symptom duration longer than 5 days. [11]

Non-specific prodrome of malaise, weight loss, low-grade fever, and gradual onset of headache over 1–2 weeks; followed by worsening headache, vomiting, and confusion, leading to coma and death if untreated.[11]

SIGNS:

Variable degrees of neck stiffness; cranial nerve palsies (VI > III > IV > VII) develop as disease progresses and confusion and coma deepen; monoplegia, hemiplegia, or paraplegia in about 20% of cases.[11]

DIAGNOSIS:

Available TBM diagnostic tests are inadequate, for the most part due to low bacillary loads in CSF. CSF Ziehl–Neelsen staining is rapid, but sensitivity is poor (approximately 10%–20%). Mycobacterial culture is more sensitive (approximately 60%–70%) but culture often takes 2 weeks [11]. Given the lack of adequate TBM diagnostics, Xpert appeals as a new diagnostic tool. Even the best current testing may miss up to 30% of cases. Clinicians are often left to treat empirically with prolonged regimens with significant side effects or risk a missed case that would result in death. Diagnosis usually relies on clinical evidence, which combines supportive clinical, laboratory, and radiological findings. an international tuberculous meningitis workshop took place in Cape Town, South Africa, in May, 2009, to establish a consensus case definition for tuberculous meningitis for use in future clinical research.[10]

TUBERCULOUS MENINGITIS CLASSIFICATION [10]

Clinical entry criteria: Symptoms and signs of meningitis including one or more of the following: headache, irritability, vomiting, fever, neck stiff ness, convulsions, focal neurological deficits, altered consciousness, or lethargy.

1. Definite tuberculous meningitis

• Patients should fulfill criterion A or B:

A) Clinical entry criteria plus one or more of the following: acid-fast bacilli seen in the CSF; Mycobacterium tuberculosis cultured from the CSF; or a CSF positive commercial nucleic acid amplification test.

B) Acid-fast bacilli seen in the context of histological changes consistent with tuberculosis in the brain or spinal cord with suggestive symptoms or signs and CSF changes, or visible meningitis (on autopsy).

2. Probable tuberculous meningitis

• Clinical entry criteria plus a total diagnostic score of 10 or more points (when cerebral imaging is not available) or 12 or more points (when cerebral imaging is available) plus exclusion of alternative diagnoses. At least 2 points should either come from CSF or cerebral imaging criteria.

3.Possible tuberculous meningitis

• Clinical entry criteria plus a total diagnostic score of 6–9 points (when cerebral imaging is not available) or 6–11 points (when cerebral imaging is available) plus exclusion of alternative diagnoses. Possible tuberculosis cannot be diagnosed or excluded without doing a lumbar puncture or cerebral imaging.

Not tuberculous meningitis

• Alternative diagnosis established, without a definitive diagnosis of tuberculous meningitis or other convincing signs of dual disease.

Diagnostic criteria for classification of definite, probable, possible, and not tuberculous meningitis.

A. Clinical criteria (Maximum category score=6)

1. Symptom duration of more than 5 days - 4 points

2.Systemic symptoms suggestive of tuberculosis (one or more of the following): weight loss (or poor weight gain in children), night sweats, or persistent cough for more than 2 weeks - 2 points

3. History of recent (within past year) close contact with an individual with pulmonary tuberculosis or a positive TST or IGRA (only in children 50%) – 2 points

4. Focal neurological deficit (excluding cranial nerve palsies) – 1 point

5. Cranial nerve palsy -1 point

6. Altered consciousness -1 point

B. CSF criteria (Maximum category score=4)

1. Clear appearance - 1point

2. Cells: 10–500 per µl – 1 point

3. Lymphocytic predominance (>50%) – 1 point

4. Protein concentration greater than 1 g/L- 1 point

5. CSF to plasma glucose ratio of less than 50% or an absolute CSF glucose concentration less than 2·2mmol/L - 1 point

C. Cerebral imaging criteria (Maximum category score=6)

1. Hydrocephalus -1 point

2. Basal meningeal enhancement – 2 points

3. Tuberculoma – 2 points

4. Infarct – 1 point

5.Pre-contrast basal hyperdensity -2 points

D. Evidence of tuberculosis elsewhere (Maximum category score=4)

1.Chest radiograph suggestive of active tuberculosis: signs of tuberculosis=2; miliary tuberculosis=4 - 2/4 points

2.CT/ MRI/ ultrasound evidence for tuberculosis outside the CNS -2 points

3.AFB identified or Mycobacterium tuberculosis cultured from another source—ie, sputum, lymph node, gastric washing, urine, blood culture – 4 points

4. Positive commercial M tuberculosis NAAT from extra-neural specimen - 4 points

Exclusion of alternative diagnoses

An alternative diagnosis must be confirmed microbiologically (by stain, culture, or NAAT when appropriate), serologically (eg, syphilis), or histo-pathologically (eg, lymphoma). The list of alternative diagnoses that should be considered, dependent upon age, immune status, and geographical region, include: pyogenic bacterial meningitis, cryptococcal meningitis, syphilitic meningitis, viral meningo-encephalitis, cerebral malaria, parasitic or eosinophilic meningitis (Angiostrongylus cantonesis, Gnathostoma spinigerum, toxocariasis, cysticercosis), cerebral toxoplasmosis and bacterial brain abscess (space-occupying lesion on cerebral imaging)and malignancy (eg, lymphoma)

ROLE OF ADA

The measurement of adenosine deaminase (ADA) level in CSF has been demonstrated to be a suitable test for TBM diagnosis.

Pormohammad et al. performed a meta-analysis in 2017 to evaluate the accuracy of ADA. Twenty studies were eligible for inclusion within the meta-analysis. The pooled sensitivity and specificity for TBM diagnosis hallmarks were 89% and 91% respectively [12]. It was shown that ADA testing has a relatively high accuracy for TBM diagnosis. This test is also recommended for TBM diagnosis in IDSA/CDC/ATS (Infectious Disease Society of America/Control Disease Center/American Thoracic Society) [13].

MANAGEMENT

Antitubercular treatment before the onset of coma is the strongest predictor of survival from TBM. The current guidelines recommend treatment with four anti-TB drugs for at least the first 2 months of therapy, followed by treatment with three drugs (rifampicin, isoniazid and ethambutol) for an additional 7 to 10 months. These recommendations are based on data from pulmonary TB and do not take into account the differential ability of anti-TB drugs to penetrate the brain.[14]

Rifampicin is a key component in TB treatment, but concentrations of the drug in CSF are less than 30% of the concentration in plasma.

Recent randomized comparison of higher-dose intravenous rifampicin (approximately 13 mg per kilogram per day) versus a standard oral dose (10 mg per kilogram per day) in 60 Indonesian adults with TBM showed that mortality among patients who received the higher intravenous dose was 50% lower than that among patients who received the standard dose [15]

Heemskerk et al. compared a standard, 9-month anti-TB regimen (which included 10 mg of rifampin per kilogram of body weight per day) with an intensified regimen that included higher-dose rifampin (15 mg per kilogram per day) and levofloxacin (20 mg per kilogram per day) for the first 8 weeks of treatment in 819 Vietnamese patients with TBM [14]. Intensified anti-TB treatment was not associated with a higher rate of survival among patients with TBM than standard treatment [14] Nevertheless, there is today good evidence from preclinical, clinical, and modeling studies to support the use of high-dose rifampicin in TBM, likely to be at least 30 mg/kg. Higher-dose isoniazid could be beneficial, especially in rapid acetylators. The role of other first- and second-line drugs is unclear, but observational data suggest that linezolid, which has good brain penetration, may be beneficial. [16]

Thwaites et all in 2011 randomly assigned adults with TBM to treatment with standard antituberculosis treatment alone or in combination with ciprofloxacin (750 mg/12 h), levofloxacin (500 mg/12 h), or gatifloxacin (400 mg/24 h) for the first 60 days of therapy and it was found that Fluoroquinolones add antituberculosis activity to the standard treatment regimen, but to improve outcomes of TBM, they must be started early, before the onset of coma. [17]

Adjunctive treatment

Intracerebral inflammation has long been recognized as an important determinant of TBM outcome. A 2016 Cochrane systematic review and meta-analysis of all relevant published trials concluded that corticosteroids increase survival in HIV-1-negative children and adults with TBM, but that the benefit of corticosteroids was uncertain in individuals co-infected with HIV-1, and was not found to reduce long-

term neurological disability. Inflammatory complications can occur weeks or even months after the start of antituberculosis chemotherapy for TBM, when initial corticosteroid treatment has been reduced or stopped.[18]

Supportive management

Supportive therapies include optimization of patient position for CSF and cerebral venous drainage, control of hyperthermia and hypotension, management of seizures, and appropriate mechanical ventilation. Drug therapies include acetazolamide for reducing CSF production, adjunctive anti-inflammatory therapy and anti-TB chemotherapy. Coma in TBM is associated with raised intracranial pressure. Hydrocephalus but also cerebral edema and tuberculoma formation can elevate intracranial pressure (ICP) in patients with TBM. A sustained ICP of more than 20 mm Hg is considered abnormal in adults.[19] Hydrocephalus is the most common cause of raised ICP. In the majority (80%) of patients with TBM, communicating hydrocephalus is observed, caused by exudates in the basal cisterns and resulting in disruption of CSF flow. In that case, reasonable evidence suggests that hydrocephalus in TBM can be treated medically with repeated lumbar punctures. Non-communicating hydrocephalus, caused by obstruction at the level of the cerebral aqueduct and/or the outlet foramina of the fourth ventricle, is less common in this population, and should be treated with ventriculoperitoneal shunting or endoscopic third ventriculostomy. [15] In fact, no optimum management strategy for TB hydrocephalus is universally accepted.

VIRAL MENINGITIS/ MENINGO-ENCEPHALITIS

The term aseptic meningitis is used to describe meninges inflammation that is caused by pathogens other than pus-producing bacteria. Viral meningitis is the most common type of aseptic meningitis and usually affects young children [3].

Encephalitis is an inflammation of the parenchyma of the brain. Viral encephalitis implies that a virus directly invades and replicates in cells

within the brain. The term “encephalitis” also indicates a clinical syndrome arising from infection and inflammation in the parenchyma, rather than in the leptomeninges. When both the leptomeninges and brain parenchyma are involved, the term “meningoencephalitis” is preferred.[9]. The epidemiology of viral meningitis in adults worldwide has been discussed in few studies mentioned below.

1. In a study done in England between years 2011 and 2014, 57% (638) cases were found to be due to Viral Meningo encephalitis and Non-Polio Enteroviruses were the most common organism isolated. [20]
2. In a study done in Greece, between 2003-2006, 44.4% (36) cases were found to be due to Viral etiology among patients with CNS infections where EV, VZV, and HSV-2 were among commonest etiologies.[21]
3. In South Korea, a study done between 2008-2013, 54.2% (96) patients of Viral Meningo encephalitis were documented among total Encephalitis cases.[22]
4. Similarly in Italy, a study was done between 2002-2006 where cases of meningitis were studied where viral etiology was found in 162(80%) patients.[23]
5. In Finland, a study conducted between 1999 to 2003 showed that incidence of Viral Meningoencephalitis was 66%.[24]

The most recently detected viral agent causing meningitis was reported in a case study of a 24-year-old male from Japan who had aseptic meningitis caused by SARS-CoV-2, which was demonstrated by performing an RT-PCR test on a CSF sample from the patient. This case report indicates the possible neuro-invasive potential of SARS-CoV-2 and its potential to cause CNS complications such as meningitis. [25]

A study done in Denmark showed similar findings, with non-polio enteroviruses being the most common causative agent. They also showed that the incidence of aseptic meningitis decreases with

increasing age (58.7 per 100,000 after birth, 38.7 per 100,000 in 6-month-old infants, and 15.6 per 100,000 in 5-year-old children) [26]
Worldwide, JE is the single largest cause of viral encephalitis (VE) [27]
The most common sporadic infectious encephalitis is herpes simplex virus encephalitis (HSVE).[28]

DIAGNOSIS

In viral meningitis, the WBC count typically ranges between 80 and 100 cells/µl (pleocytosis), with a large proportion of lymphocytes (>80%) [29]. Also, glucose and protein levels usually remain normal [30]. Pleocytosis generally is considered an important criterion for diagnosis of viral meningitis. However, a number of studies have shown that pleocytosis is often not observed in EV meningitis cases involving infants (38%) or children (39%), or when a lumbar puncture is performed at an early stage of disease [31, 32] It has been found that 25% of patients with CNS abnormalities caused by viral agents have neutrophilic pleocytosis [33]. Moreover, a study showed that 47% of study subjects with enteroviral meningitis had neutrophilic pleocytosis. The gold standard tool for diagnosis of viral meningitis is the polymerase chain reaction (PCR), which detects and quantifies viral DNA or RNA in the patient's CSF [34]. A study of EV-positive meningitis cases in pediatric patients showed that testing of CSF samples by real-time PCR (RTPCR) resulted in 100% sensitivity, compared to only 38% sensitivity when using viral culture. Similarly, in a study of HPV-positive meningitis cases, 100% sensitivity was achieved with RT-PCR, while viral culture failed to detect the virus [35] However, since obtaining CSF is difficult, alternative specimens, such as blood, throat or nasal swabs, or stool samples might be used instead, particularly in cases of suspected EV infections [36]. On the other hand, diagnosis of mumps virus, herpesvirus, arboviruses, and human immune deficiency virus (HIV) infections can be achieved using serological assays, although the results might be negative at early stages of the disease. Hence, a second sample should be obtained after two weeks and tested again for the presence of viral agents [4]. Next-generation sequencing (NGS) was recently used in a number of studies to detect a

wide variety of pathogens, including viruses, the possible application of this technique as a diagnostic tool in meningitis cases is not yet clear. MRI and EEG should also be performed.

CNS INFECTING VIRUSES

I. Family Picornaviridae

a. *Enteroviruses (EVs)*:

In most cases, the virus enters the body through the oral-fecal route. Various studies have shown that EVs infect different neural cells, such as glia cells and astrocytes [37], the virus could reach the bloodstream by infecting immune cells and cause secondary infections at other sites, such as the heart and CNS [38]. This dissemination of the virus leads to different complications, such as meningitis and encephalitis [39].

Coxsackievirus B, a member of the species Enterovirus B, is known to cause meningitis and other neurological disorders [40].

b.Echoviruses, such as E-6, E-9, E-11, E-13, E-19, and E-30 also belong to the species Enterovirus B and are commonly known to be associated with viral meningitis in children [41].

c. Enterovirus D68 (EV-D68) is the most common member of the species Enterovirus D found to be associated with the development of aseptic meningitis. EV-D68 can cause CNS complications in children, commonly meningitis and flaccid paralysis [42]

II. Family Herpesviridae

Major members of the family Herpesviridae that can cause meningitis include human herpesvirus 1 and 2 (HSV-1 and HSV-2), varicella-zoster virus (VZV), and Epstein-Barr virus (EBV) [43]. The major neurological consequences of HSV are encephalitis and meningitis. All of these viruses are known to cause meningitis and can establish a latent infection in neurons, which can be reactivated [43]. Herpes simplex type 1 virus (HSV-1) is the usual virus identified, with more than 90% of cases related to HSV-1. It tends to cause clinical signs of focal cortical neurologic deficits including hemiparesis, aphasia, and seizures. HSE

commonly involves limbic parts of the brain, which can lead to prominent behavioral changes at the beginning of the illness before the patient's level of consciousness is depressed. Focal or generalized seizures are particularly common when encephalitis affects the cerebral cortex, especially the temporal lobes.[9]

III. Family Orthomyxoviridae

Human influenza viruses often cause pneumonia and acute respiratory distress syndrome (ARDS) [44], but on rare occasions, they can cause extra-respiratory illnesses, including meningitis [44], encephalitis [45], meningoencephalitis, and myelitis [46]

IV.Arboviruses

Several arboviruses of different families have been associated with CNS infection and development of meningitis. These include members of the families Flaviviridae (e.g., West Nile virus [WNV]), Togaviridae (e.g., Eastern equine encephalitis virus), and Peribunyaviridae (e.g., La Crosse virus). Most of these viruses are zoonotic and are transmitted to humans through arthropods such as mosquitoes, ticks, and sandfies. The most important arbovirus associated with CNS complications is WNV [47].

MANAGEMENT

In a study done in the UK, researchers found that the median length of hospital stay was 4 days for patients with viral meningitis and nine days for those under antiviral therapy. They also concluded that delays in performing lumbar puncture and unnecessary treatments were associated with prolonged hospital stays and long-term morbidity.[20]

No specific treatment is prescribed for aseptic meningitis cases, and supportive medications are usually given to minimize disease complications such as fever and headache, and full recovery takes from 5 to 14 days in the majority of cases [4]. Regarding HSV treatment, one study showed that antiviral therapy in immunocompromised patients with HSV-induced meningitis should be started immediately and that any delay in the administration of treatment can result in the development of

adverse complications [48]. The use of acyclovir against HSV-2-induced meningitis was also evaluated, with better outcomes observed in treated patients. However, one patient developed concentration difficulties as a meningitis symptom that lasted for approximately three months [49]. Recently, a promising drug called psoromic acid (a bioactive lichen-derived compound) has been found to inhibit the replication of both HSV-1 and HSV-2 by inhibiting proteases and DNA polymerases, which makes it a possible drug for treating meningitis caused by HSV [50]. Valacyclovir was also tested in clinical trials for its antiviral suppression ability on recurrence of meningitis. However, treatment with valacyclovir (twice daily) did not prevent recurrent meningitis and was not recommended for this purpose [51,52]. For HSV associated encephalitis, before acyclovir (and the drug used to treat it previously, vidarabine), 70% of patients died of infection.[53] Even using acyclovir, which is the current therapy, mortality is still 15%, and fewer than 20% of patients are able to return to full-time employment after treatment, often because of cognitive deficits that persist. Multicenter prospective trial results emphasize that early treatment affects outcome.[54] When HSE is suspected in the acute setting by the presence of focal signs or symptoms, early empirical treatment is recommended even while the diagnostic evaluation is proceeding. The current therapy-of-choice treatment is intravenous acyclovir (10 mg/kg every 8 hours for 14–21 days). In UK and France, Multicenter trial of dexamethasone in herpes simplex virus encephalitis are underway. Moreover, V-7404 (viral polymerase inhibitor) and DAS181 (sialidase) are newly developed drugs against EV-D68 [55,56]. A recent systematic analysis showed that most viral meningitis cases have a good long-term clinical outcome and that patients with viral meningitis have a better outcome after hospital discharge than patients with viral encephalitis [57].

ACUTE PYOGENIC MENINGITIS

Four main causes of acute bacterial meningitis:

- *Neisseria meningitidis* (meningococcus)
- *Streptococcus pneumoniae* (pneumococcus)

- *Haemophilus influenzae*
- *Streptococcus agalactiae* (group B streptococcus)

These bacteria are responsible for more than half of the deaths from meningitis globally and they cause other severe diseases like sepsis and pneumonia. Post introduction of Hib and S. pneumoniae vaccines, high-income nations have witnessed a significant reduction in bacterial meningitis caused by these bacteria. However, the scenario is different in low and middle-income countries (LMICs), where bacterial meningitis continues to be a major concern. Inadequate vaccine coverage or lack of availability of these vaccines in the national immunization programmes are some of the factors responsible for this menace.[58]

In 2021, Canna J. Ghia and Gautam S. Rambhad did a systematic literature review on the prevalence and etiology of meningitis among critically ill and hospitalized patients in India [59]. The summary of all adult group studies is mentioned below with study references.

TABLE 1: VARIOUS STUDIES CONDUCTED FOR ACUTE PYOGENIC MENINGITIS IN ADULTS

Author name	Study period	Study place	Study participants	Age group	Overall prevalence
Gangane and Doddamani [60]	2010–2012	Karnataka	304 CSF samples with suspected meningitis, 104 cases of confirmed meningitis	>60years	Bacterial meningitis: 78.85% Fungal meningitis: 9.62% Acid-fast bacilli meningitis: 10.58%
Mishra et al.[61]	2011–2012 Lucknow		235 critically ill neurological patients 76 patients with CNS infection	Median age: 37.5 (4–75years	• TB meningitis: 47% • Pyogenic meningitis: 11% • Fungal meningitis: 5%
Yerramilli et al.[62]	2012–2014	Hyderabad	147 cases of meningitis		Viral/aseptic meningitis: 39% Tuberculous meningitis: 28% Pyogenic meningitis: 28% ,Fungal meningitis: 3% (4 cases
Verghese et al.[63]	2008–2016	Vellore, Tamil Nadu	830 invasive pneumococcal isolates	All age groups	20.1% (167 cases) Bacterial
Thomas et al.[64]	1993–2008	Vellore	1037 patients with suspected invasive bacterial infection	>18years	Meningitis: 34.3% Bacterial
Jayaraman et al.[65]	2007–2017	Vellore	408 adults with IPD	>18years	Meningitis: 24.3% Bacterial

Kadam et al..[66]	2011–2012	Pune, Maharashtra	208 HIV patients	40years	Cryptococcal meningitis: 19% Fungal
Wadia et al.[67]	NS	Pune	1527 HIV-positive subjects 457 HIV-positive subjects with neurological complications		Prevalence of meningitis: 17.88% • Cryptococcal meningitis: 67.44% • Tuberculous meningitis: 18.60%
Mathur and Devesh4[68]	2006–2011	Jaipur, Rajasthan	: 40 20 HIV patients with neurological complications	25–45years	Cryptococcal meningitis: 20% Tuberculous meningitis: 20% Pyogenic meningitis: 5%,Candida meningitis: 5%
Teja et al.[69]]	NS/11 years	Hyderabad	1606 hospitalized patients with HIV and neurological manifestation	36 years	Meningitis: 39.4% • Tuberculous: 25.06% • Cryptococcal: 10.95% • Pyogenic: 1.95%
Nair et al.[70]	2005–2006	New Delhi	380 suspected cases of meningococcal disease	45years	8.68% (33 cases) Bacterial
Bhagawati et al.[71]	2009–2010	Assam	316 CSF samples	0–70years	16.14% Bacterial and fungal

The commonest cause of pyogenic meningitis in these studies was S. pneumoniae, across different regions of India, with a frequency (in percentage) ranging from 12% to 82.9% in children and 4% to 61.8% in patients of all age groups. [59]

In studies reporting data on patients belonging to all age groups, apart from S. pneumoniae, Staphylococcus aureus (seven studies; frequency range 1.8–41.38%), N. meningitidis (five studies; frequency range 1–27.59%), Klebsiella pneumoniae (five studies; frequency range 5.5–20.7%), Escherichia coli (five studies; frequency range 4.76–27.08%), H. influenzae (four studies; frequency range 1.8– 19.4%) etc. were the other causative pathogens.

DENGUE ENCEPHALOPATHY AND ENCEPHALITIS

Dengue infection is widespread in the tropics. The incidence of neurological manifestations in patients diagnosed with dengue hemorrhagic fever has been documented to vary from 3% in prospective dengue hemorrhagic fever studies to 25% in retrospective studies. [72]

Encephalitis is one of the common manifestations of dengue neurological disease. Encephalopathy is attributed to: cerebral oedema; anoxia; hemorrhage; hyponatremia; liver or kidney failure; micro capillary hemorrhage; or release of toxic substance. [73,74] Cam *et al.* noted 27 cases of DHF developing encephalopathy. Of these 26 were comatose, 21 had convulsions and one had hemiplegia. No abnormalities were noted on MRI in four of 18 patients with encephalopathy. Other lesions noted on MRI were oedema; scattered focal lesions; cerebral oedema; and, in one, cerebral hemorrhage.[75]

In a study held in 2012 in CMC Hospital, Ludhiana, Punjab, Koshy et all [76] Neurological manifestations were noted in 21 (263%) of the 799 patients who were diagnosed with dengue virus infection. There were 19 men and the mean age was 33.77 ± 13.94 years. Three of the 21

patients with neurological symptoms died. The neurological diagnoses for these patients were encephalopathy, ADEM and intracranial hemorrhage, respectively.

SCRUB TYPHUS RELATED ENCEPHALOPATHY

CNS involvement is a known complication of scrub typhus which ranges from aseptic meningitis to frank meningoencephalitis.[77] Many studies in India and in other countries found that meningoencephalitis is a most common neurological complication of scrub typhus. A study done by Rana et al. [78] found that the most common neurological manifestation was meningoencephalitis (40%). A cross-sectional study on 37 patients published by Mishra et al. [79] found two-thirds of patients with scrub typhus had neurological involvement manifesting as meningoencephalitis, encephalitis, or encephalopathy, but cerebrospinal fluid findings can mimic tuberculous meningitis and viral meningoencephalitis.[77] In a Korean study, 89 patients with severe complications and 119 without severe complications due to scrub typhus were evaluated. In the group with severe scrub typhus, 23 (11.3%) patients had meningoencephalitis.[80] Scrub typhus as a cause of ADEM is extremely rare, pathophysiology is obscure, but it has been postulated to result from an autoimmune response to myelin basic protein triggered by infection as in our cases it may be due to cross reactivity of IgM antibodies to myelin protein.[80]Sardana et all also did a study in 2020 on neurological manifestations of scrub typhus and found that Meningoencephalitis was the most common encountered symptom in our study along with rare complications such as ADEM and cerebral venous thrombosis.[81]

NEUROLOGICAL MANIFESTATIONS OF LEPTOSPIROSIS

Leptospirosis is caused by the spirochete bacterium L. interrogans. The infection is transmitted to humans when water contaminated by the urine of an infected animal (typically rats or mice) comes into contact with damaged skin, eyes, or mucous membrane.[82]. Aseptic meningitis is a common feature of leptospirosis but severe symptomatic neurological

involvement is rarely observed. Central and peripheral neurological presentations have, however, been described: meningoencephalitis, encephalitis (altered sensorium, delirium, coma, psychosis, convulsions, hemiplegia, cerebellar disorder), acute disseminated encephalomyelitis, intracerebral bleed, spinal extradural hematoma, myelopathy, myeloradiculopathy, Guillain-Barré syndrome (weakness in the lower limbs, areflexia, paresthesia), neuropathies (mononeuritis, facial palsy), and myositis.[82,83] Convulsions and altered sensorium are the most common presentations.

In 2006, Mathew et all did a study where a total of 31 patients of neuroleptospirosis were treated during the five-year period. Acute fever with chills and rigors, headache and vomiting were the presenting manifestations; 25 patients (81%) had altered sensorium for a period ranging from 1- 8 days, four (12.9%) being deeply comatose. Eleven (35.5%) had acute symptomatic seizures at the time of presentation. Only three patients had localizing deficits. CT scan was normal in 18 of 27 (67%), while 7 (26%) had diffuse cerebral oedema. CSF pleocytosis with lymphocytic predominance (mean 50 cells/microliter) and elevated protein levels (mean 115.5 +/- 67.5 mg %) were noted. Leptospira antibody was detected in serum of all, and 5 of 22 in CSF samples. Eight patients (26%) succumbed. Deep altered sensorium at presentation and raised CSF protein were two poor prognostic indicators. Pathological study of brain in five cases revealed encephalitic features and in addition immune mediated acute disseminated encephalomyelitis (ADEM) like pathology in two cases.[84]

SEPSIS ASSOCIATED ENCEPHALOPATHY

Overall global incidence is approximately 31 million sepsis cases per year [85,86]. Sepsis-associated encephalopathy (SAE) is one of the most common complications during the acute phase and in later stages after surviving sepsis. It is defined by a diffuse cerebral dysfunction due to the dysregulated host response and absence of a direct central nervous system (CNS) infection. SAE symptoms may already be present before

sepsis criteria are fulfilled. Symptoms in the acute stage range from sickness behavior and delirium to coma and may later result in long-term cognitive impairment [87,88]. Among others, symptoms include agitation, hallucinations, reduced concentration, and alteration of the sleep–wake cycle [87]. Depending on the disease severity, patients may become somnolent or even comatose. Earlier studies have already suggested a high association of SAE and mortality of sepsis patients by emphasizing mortality rates over 60% in these patients [89]. Even mild alterations of the mental status (GCS of 13–14) seem to have prognostic potential towards a worse outcome in sepsis [90]. Delirium due to SAE correlates with the development of long-term cognitive dysfunction following hospital discharge [91] As SAE is a diagnosis of exclusion, other causes of encephalopathy (e.g., metabolic changes, drug intoxication, structural brain lesions, cerebrovascular events, encephalitis, meningitis, and non-convulsive status epilepticus) need to be ruled out in sepsis patients [92]. The mortality rate of sepsis patients increases from 26% to 49% with SAE and is associated with higher values of the GCS, sequential organ failure assessment score (SOFA), and the APACHE II score [90,93,94]. At hospital discharge, almost 45% of sepsis survivors show symptoms of long-term cognitive dysfunction [95]. However, an exact evaluation of incidence, prevalence, and mortality is restricted due to lack of definite SAE diagnosis criteria in these studies and due to the variable clinical presentation of SAE.

CEREBRAL IMAGINING:

Despite severe symptoms, in 52% of SAE patients, cerebral imaging is unremarkable, especially in acute stages. In a prospective observational study, MRI scans in acute stages of SAE identified ischemic lesions (29%) as the most common pathological findings [96]. These lesions are displayed in diffusion-weighted imaging and represent cytotoxic edema usually caused by ischemia, hypoxia, or vasogenic edema and may indicate circulatory impairment (e.g., impaired macro- and micro-circulation).[97] In addition, ischemic stroke was independently associated with increased mortality and poor neurologic outcome.[98] In

approximately 21% of sepsis patients, leukoencephalopathy can be found in MRI, which might be a possible marker of BBB leakage [98]. In some cases, signs of a posterior reversible encephalopathy syndrome can be detected.[99]

EEG:

Several case series and small studies describe a variable prevalence of electroencephalography EEG) abnormalities (ranging from 12% to 100%), e.g., appearance of theta and delta waves [98,100,101]. This high range of abnormal EEG findings might be due to the small sample size and heterogeneity of the study population. Severe EEG abnormalities with periodic and rhythmic discharges (e.g., triphasic waves, frontal intermittent rhythmic delta activity, general periodic discharges) may indicate severe SAE. Sometimes, epileptiform discharges (e.g., periodic lateralized, bilateral independent lateralized) can be recorded [98,100,101,102]. In sepsis patients with bacteremia but no detectable cognitive deficits, abnormalities in EEG could be demonstrated in even 50% of cases.[101] Thus, EEG as a non-invasive investigation tool is helpful to assess the severity of SAE, as it reflects the degree of encephalopathy in general. Absence of EEG modulation, a delta-predominant background, and periodic discharges might be independent predictors of mortality and were also associated with the occurrence of delirium in sepsis patients [103,104]. In addition, seizures occur in almost 10%–20% of sepsis patients, most commonly non-convulsive seizures.[101,102] However, it is important to note that none of these EEG abnormalities are specific for SAE and can also widely occur in encephalopathy of other etiology.

CSF:

Clinical routine laboratory testing of CSF is restricted to the exclusion of other etiologies of encephalopathy, such as meningitis and encephalitis. Findings in SAE are limited to a slight protein increase as a sign of local inflammation or impairment of BBB without specific intrathecal immunoglobulin synthesis.

POST COVID ENCEPHALOPATHY:

The pathogenesis of encephalopathy associated with COVID-19 may be multifactorial. Postulated mechanisms include hypoxic/metabolic changes produced by the intense inflammatory response due to cytokine storm and neurotropism. Cytokine storm causes hypoxia and metabolic insults that result in global dysfunction of the brain. Altered consciousness, ranging from mild confusion, delirium, to deep coma, are some of the cardinal clinical features. The most common imaging finding on MRI T2-weighted fluid-attenuated inversion recovery (MRI T2/FLAIR) includes symmetric, multifocal lesions with invariable thalamic involvement. Other commonly involved locations include the brainstem, cerebral white matter, cortical and subcortical white matter, and cerebellum. In a few case reports, cerebrospinal fluid (CSF) analysis has shown the presence of the virus. Management of encephalopathy in COVID-19 patients involves supportive care including supplemental oxygen therapy and immune modulators.

Shah et al reported a case of a 67-year-old male who was admitted to the hospital for the management of COVID-19 pneumonia. Due to worsening hypoxia, the patient was transferred to ICU and was intubated. On examination, he was aphasic and noted to have right-sided hemiplegia with left-sided hemiparesis on day 4. CT scan of the head without contrast and MRI findings were suggestive of acute necrotizing encephalopathy secondary to intracranial cytokine storm caused by viral infection. The patient was treated with intravenous immunoglobulin (IVIG), and high dose corticosteroids, with clinical improvement in the right-sided hemiparesis on day 5. A repeat MRI brain revealed decreased edema.[105]

Following are a few more studies which have been conducted earlier for patients with Acute Febrile Encephalopathy.

In 2001, Panagariya et al did a study on Herpes simplex encephalitis in North West India and found that Main clinical features were seizures (89%) and altered sensorium (100%). CSF examination was abnormal in 25 (90%) cases with (64%) patients. CT scan showed asymmetric frontotemporal lesions with or without hemorrhage in 18 (64%) cases. MRI was fairly characteristic with bilateral asymmetric frontotemporal lesions in all the 16 patients, in whom it was done. Out of the 28 patients, 18 reported within one week of the onset of symptoms. Of these, 13 patients had altered sensorium (grade I-II) and the disease was acute. Acyclovir was given to them immediately. 23% patients showed good recovery (Gr.-5), and 23% patients died. The remaining 5 patients in this group were stuporosed / comatosed on admission. They were given steroids along with acyclovir. Three patients (60%) survived with moderate residual deficits (Gr.-4) and two (40%) died. Out of 10 patients who reported after one week of onset of the first symptom, 3 were stuporosed/comatosed (grade III-IV) and they died early in the course of the disease, even after treatment with acyclovir and steroids. Remaining 7 patients were treated with acyclovir alone. Four of them (57%) survived with moderate residual deficits (Gr.-4), while 3 (43%) patients died.[106]

In 2005, Bansal et al conducted a study on “To study the etiology and clinical profile of non-traumatic coma in children and to determine the clinical signs predictive of outcome.” And concluded that etiology of coma in 60% cases was CNS infection (tuberculous meningitis-19, encephalitis-18, bacterial meningitis-16, others-7); other causes were toxic-metabolic conditions (19%), status epilepticus (10%), intracranial bleed (7%), and miscellaneous (4%). 65 children survived, 11 were normal, 14 had mild disability, 21 had moderate disability and 14 were severely disabled and dependent. Survival was significantly better in patients with CNS infection (63%) as compared to those with toxic-metabolic causes (27%) and intracranial bleed.[107]

In 2007, Kumar et al conducted a study titled Dengue encephalopathy in children in Northern India: Clinical features and comparison with non dengue and reported that out of a total of 265 patients of AFE were enrolled over a 2 year period, HI test was positive in 15/49 (30.6%) and IgM in 52/238 (21.8%) patients thus tested. A total of 62 patients were positive for dengue antibodies by either test. Real time PCR assay for dengue virus genome was positive in 28/42 (69%) tested — 21/29 (72.4%) in CSF and 9/15 (60%) in serum. A total of 39 patients met the criteria for definite dengue infection of which only 2 fulfilled the WHO criteria for DHF. Comparing DE and non DE, rash, bleeding, swelling over body, and hepatomegaly were significantly more common and meningeal signs less frequent in DE. Mean platelet counts and serum albumen were lower and liver enzymes and INR were significantly higher in DE.[108]

In 2008, Karmakar et al conducted a study of A study of acute febrile encephalopathy with special reference to viral etiology on 151 patients with mean age of 3.21 ± 2.9 (range of mth-13 years) and male: female ratio of 1.71: 1. A diagnosis other than viral encephalitis was reached in 94 patients (62.3 %). Pyogenic meningitis was the most frequent diagnosis 51(33.8 %) followed by tuberculous meningitis 12 (7.9 %), and cerebral malaria 8 (5.2 %) in the patient group of non-viral causes. Fifty-seven cases (37.3%) were suspected as viral encephalitis and mean age of the cases suspected as viral encephalitis was 2.8 ± 2.9 (Range 1 mth-10 yrs) with male: female ratio of 1.28: 1. Etiological diagnosis was reached or considered probable in 41 (72%) cases out of the suspected patients. The most common etiological agent identified was enterovirus 71 in 20 patients (35.1 %). The other viruses identified were mumps in 6 (10.5%), Japanese encephalitis in 5 (8.7%), and measles in 4 (7%) cases. MRI brain was done in 39 patients and was abnormal in 14 patients. Out of 57 cases of suspected viral encephalitis 10 patients expired within 48 hours, 2 > 48 hours and 19 patients had significant neurological sequels at discharge.[109]

In 2014, Ashish Bhalla et al conducted a study on “Acute febrile encephalopathy in adults from Northwest India and found that 70% had primary CNS infection as the etiology. A total of 33% patients had meningitis, 29.9% had evidence of meningoencephalitis, and 12.7% were diagnosed as sepsis-associated encephalopathy. These were followed by cerebral malaria, leptospirosis, and brain abscess as the cause of febrile encephalopathy in adults. Among the noninfectious causes, acute disseminated encephalomyelitis, cortical venous thrombosis, and neuroleptic malignant syndrome were documented in 2.36% each. In 11% of the patients, the final diagnosis could not be made in spite of the extensive investigations. His study demonstrated that acute febrile encephalopathy in adults is a heterogeneous syndrome with primary CNS infections being the commonest etiology.[110]

In 2015, Naderi Hamiz Raza conducted a study on “Investigating the Causes of Febrile Encephalopathy in Elderly Patients Admitted to Imam Reza Hospital in Mashhad during the 2013 to 2014” and found that Infectious causes were responsible for febrile encephalopathy in 94 patients, of which 84% had an extra- cranial infection and neuro-infectious process was detected in 10%. The age of all 100 participants were 65 years and over consisting of 39 female and 61 male. Of them, only 34 patients underwent lumbar puncture, among them the cerebrospinal fluid (CSF) analysis was abnormal in 29.4%.[111]

In 2013, Anusha Deepthi et al did a study on acute febrile encephalopathy in children. In this study, AFE was higher among the age group of 6-14 years (54.7%) with most common cause being viral encephalitis (38%). Higher mortality rates were observed in CNS infections. Persistent seizures and hemiparesis were the most common causes of morbidity. GCS at initial presentation <7 was significantly associated with mortality, p value being 0.036.[112]

In 2006, ME Yeolekar, TH trivedi published an editorial on “Febrile Encephalopathy: challenges in Management” and concluded that clinician needs to consider a host of conditions of diverse origin,

depending on geographic area, seasonal trend and recent spurts. Index of clinical suspicion indeed needs to be high. Initial empirical treatment after collection of blood/ CSF is justified. Treatment based on close continuous monitoring of clinical, hemodynamic and laboratory parameters with appropriate imaging can only help salvaging the patient and minimizing sequelae in the clinical challenge posed by acute febrile encephalopathy.[113]

In November, 2016, Avinash Aggarwal, Manish Gutch et al did a study named “State of Globe Acute Febrile Encephalopathy” and concluded that, patients can make complete recovery provided the underlying cause is treated timely. Efficiency and skill are required to correctly and timely diagnose the underlying etiology. A small but significant number of patients die, and some of the patients are left with neurologic sequelae. Delayed neurologic recovery and sequelae are also well recognized. The fact that maximum mortality is seen in patients with AVE and SAE signifies that diffuse cerebral parenchymal involvement, lack of availability of specific drug for most of the viral infections, and multiorgan dysfunction in SAE possibly contribute to the overall increased mortality in these patients. Raised intracranial pressure in meningoencephalitis also contributes to mortality. Patients in whom a definitive diagnosis cannot be reached also show higher mortality.[114]

In 2015, Sukrirti Kumar et al did a study called Acute viral encephalitis clinical features and outcome: Experience from a tertiary center of North India and concluded that JE is the most common etiology identified followed by HSV encephalitis. The outcome in cases with AVE can be fatal or more disabling than other etiologies. Higher prevalence of JE is related to geographic and seasonal variations.[115]

In 2012, Nirdesh Jain et al did a case study on fatal combination of dengue shock syndrome with subdural hematoma. A 22-year-old male was admitted with high-grade fever since 1 week and headache since 3 days and unconsciousness since 1 day. History of head trauma was absent. Past history revealed that he had had dengue fever last year for

which hospital admission was not required and he had recovered within a week. On examination, the patient was cyanosed with cool extremities and blood pressure was not recordable. On abdominal examination, abdomen was distended; free fluid was present without having any organomegaly and bowel sounds were present. Neurological examination revealed no signs of meningeal irritations, a Glasgow Coma Scale (GCS) score of 4 (E1M2V1) and bilateral plantar reflex was non-elicitable. Computed tomography (CT) scan brain showed massive left parietal acute SDH obliterating left lateral ventricle with midline shift. Diagnosis of DSS was considered on clinical and investigational background. The patient was given fluid resuscitation and mechanical ventilation and, but the blood pressure was not picked up even after 3 hours of resuscitation. Subsequently, he was given vasopressor support. Next day, four units of platelets were transfused when his platelet count dropped to 52,000/mm 3. Despite all life-saving measures, he died.[116]

In 2010, J.M.K Murthy did a study on Neurological manifestations of Dengue and concluded that Encephalopathy is the most common neurological manifestation of dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS), and the pathophysiology is multi-factorial and includes cerebral edema, cerebral hemorrhage, hyponatremia, hepatic failure, renal failure and cerebral hypoxia Pathological studies in fatal cases of dengue showed nonspecific lesions, edema, vascular congestion, focal hemorrhages. In patients with dengue encephalopathy, magnetic resonance imaging (MRI) brain abnormalities included edema and scattered focal lesions, hemorrhage and cerebral edema.[117]

In 2002, PGE Kennedy and A. Chawdhary published an article on herpes simplex encephalitis and concluded that Both the mortality and morbidity of patients with HSE have been dramatically improved by the advent of the nucleoside analogue acyclovir (acycloguanosine) especially if it is initiated early, as soon as the diagnosis of HSE is

suspected Acyclovir resistance in proven HSE has not been shown to occur in immunocompetent patients, 27 although HSV resistance to acyclovir has been reported in the immunocompromised.28 Whether intravenous acyclovir treatment should be followed by the use of valacyclovir orally26 for an extended period is an issue that has yet to be resolved. Despite these remarkable advances in treatment, HSE still has an unacceptably high mortality (20–30% with acyclovir) 6, 24 and morbidity. A better outcome is seen in younger patients below the age of 30 years and in those in whom the duration of encephalitis was four days or less and the Glasgow coma score was above 6 when acyclovir was started.[118]

In 2006, VM Kothari et al did a study on tropical infections in ICU and concluded that Fever with altered mental status is produced by bacterial meningitis, Japanese B encephalitis, cerebral malarial, typhoid encephalopathy and fulminant hepatic failure due to viral hepatitis. Subtle differences in features of the organ failure exist among these infections. The diagnosis in some of these diseases is made by demonstration of antibodies in serum, and these may be negative in the first week of the illness. Hence empiric therapy for more than one disorder may be justified in a small proportion of cases. In addition to specific anti-infective therapy, management of organ dysfunction includes use of mechanical ventilation, vasopressor drugs, continuous renal replacement therapy and blood products. Timely transfer of these patients to well-equipped ICUs with experience in managing these cases can considerably decrease mortality and morbidity.[119]

MATERIALS AND METHODS

This study was conducted at Dayanand Medical College and hospital, Ludhiana. It was an observational study, using data from patients admitted in DMCH, medicine and neurology emergency, ICU's and wards who presented with acute febrile encephalopathy.

Source of Data

This was an observational prospective study to be conducted on all the patients who presented with complaints of fever accompanied or followed by altered sensorium fitting into the inclusion criteria of the study in the Department of Medical Emergency of DMCH, Ludhiana over a period of 1 and half years. Outcome of the patients was assessed at the time of discharge.

METHOD OF COLLECTION OF DATA

All patients presenting with fever with altered sensorium either at onset or following fever and willing to participate in the study after giving informed consent were included in the study. Appropriate history and clinical examination was done and routine investigations like complete hemogram, RFT, LFT, RBS, Blood C/S, WIDAL and investigations which are a part of evaluation of the cause of such presentation like Tropical fever profile, Lumbar puncture, Radio imaging was done.

SAMPLE COLLECTION:

1. Blood Sample: Under all aseptic conditions, 10-20 ml of blood was withdrawn from accessible veins and inoculated directly into culture bottles.
2. CSF sample was collected aseptically by lumbar puncture into sterile glass vials. Direct examination included wet mount preparation, India Ink preparation, Bacterial antigen and Cryptococcal antigen.

   Blood and body fluid specimens were processed in BACTEC 9240 or BacT/Alert microbial detection system, incubation was done for a maximum period of 7 days, smears were prepared from positive blood

culture bottles and Gram staining was done. The positive bottles were sub-cultured in Microbiology Department.

Samples for CSF Biochemistry and Cytology were sent in respective Departments.

3. For Tropical Fever serology, blood samples were taken in vacutainers under all aseptic conditions and processing was done as per standard kit protocol and reporting was done as Reactive or Non-Reactive.

   The work up for Tropical fever included:

   i. Dengue NS 1 Antigen
   ii. Dengue IgM
   iii. Scrub Typhus IgM
   iv. Leptospira IgM
   v. Malaria Antigen
   vi. PBF for Malaria

INCLUSION CRITERIA

- age more than 18 years
- patients/attendants who agreed to participate in the study after signing informed consent
- patients presenting with fever of less than 15 days duration which was either accompanied or followed by altered mental status. (GCS<12)
- gender: either

EXCLUSION CRITERIA

- Consent not available
- age <18 years
- cerebral vascular accident followed by fever
- any metabolic cause which was independently explaining the presentation of altered sensorium.

DURATION OF STUDY: One and half year.

STATISTICAL ANALYSIS: Data was described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations were done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

OBSERVATIONS AND RESULTS

A total of 271 patients presenting to Dayanand Medical College and Hospital with fever of onset within 15 days prior to admission and accompanied with or followed by altered sensorium were studied. A questionnaire containing history, examination, relative investigations like biochemical, pathological and microbiological serum markers, CSF analysis, neuroimaging, EEG etc of the patients was prepared and completed for all the patients.

DEMOGRAPHIC PROFILE

Most patients in our study were in the age group >60 years. The mean age of presentation was 53.71 years ±19.41 years.

TABLE 2: AGE DISTRIBUTION OF PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY

Age group (in years)	No. of cases	Percentage
18-40	78	28.7%
41-60	83	30.6%
>60	110	40.5%
Total	271	100.0%

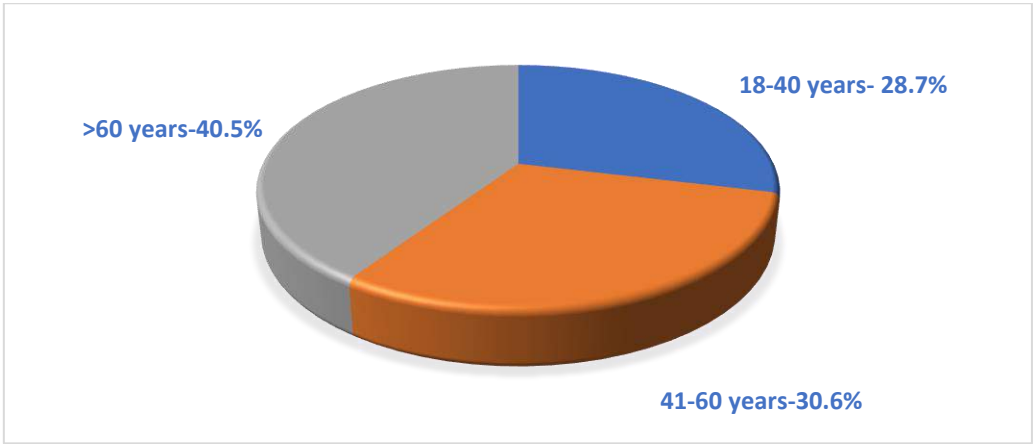


FIGURE 1: AGE DISTRIBUTION OF PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY

SEX DISTRIBUTION:

In our study, there were 105 females (38.7) and 166 males (67.3) out of 271 patients. The male:female ratio was 1.57:1

TABLE 3: SEX DISTRIBUTION OF PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY

SEX	NO. OF CASES	PERCENTAGE
F	105	38.7%
M	166	61.3%
Total	271	100.0%

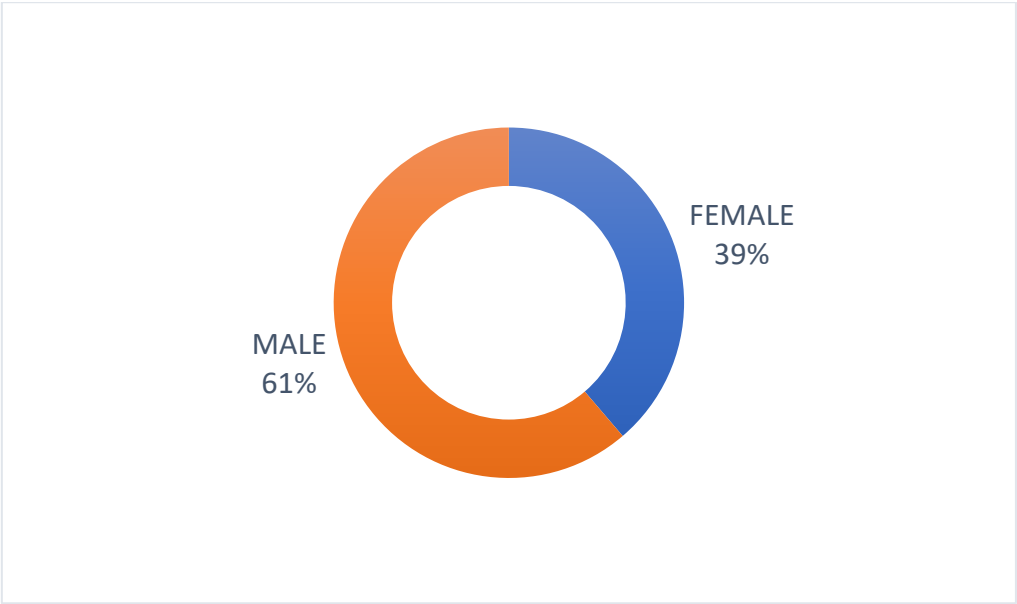


FIGURE 2: SEX DISTRIBUTION OF PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY

PRESENTING COMPLAINTS:

It was observed that in our study, all the subjects fulfilled the inclusion criteria of fever and altered sensorium. Apart from that, headache (50%), vomiting (49%), abnormal body movements (49%) were the major presenting symptoms.

TABLE 4: PRESENTING COMPLAINTS OF PATIENTS WITH AFE

CHIEF COMPLAINTS	NO. OF CASES	PERCENTAGE
FEVER	271	100.0%
ALTERED SENSORIUM	271	100.0%
HEADACHE	50	18.5%
VERTIGO	4	1.5%
BLURRING VISION	13	4.8%
ABNORMAL BODY MOVEMENTS	48	17.7%
WEAKNESS IN ANY PART OF BODY	9	3.3%
VOMITING	49	18.1%
OTHERS	75	27.7%

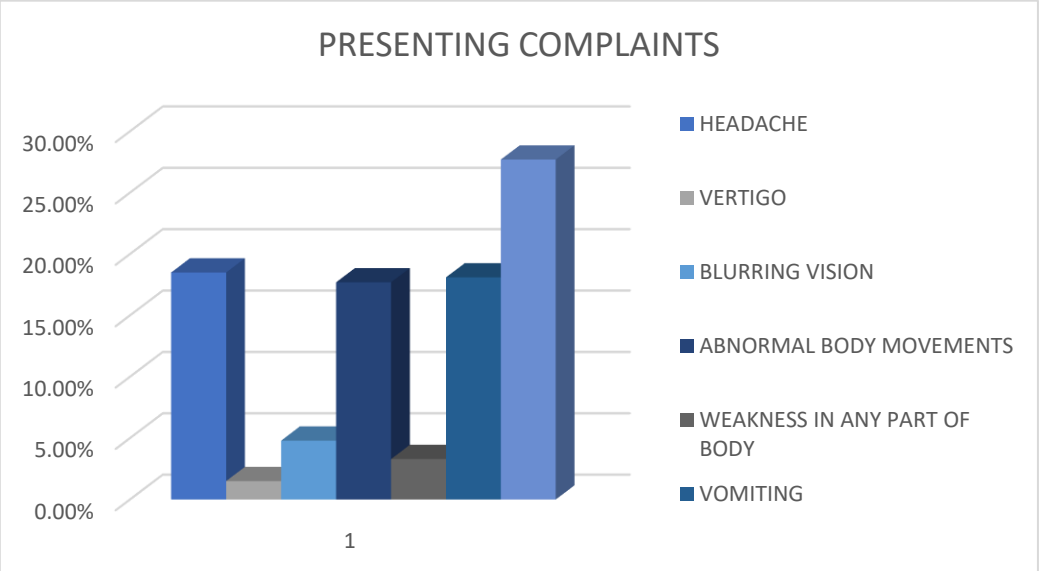


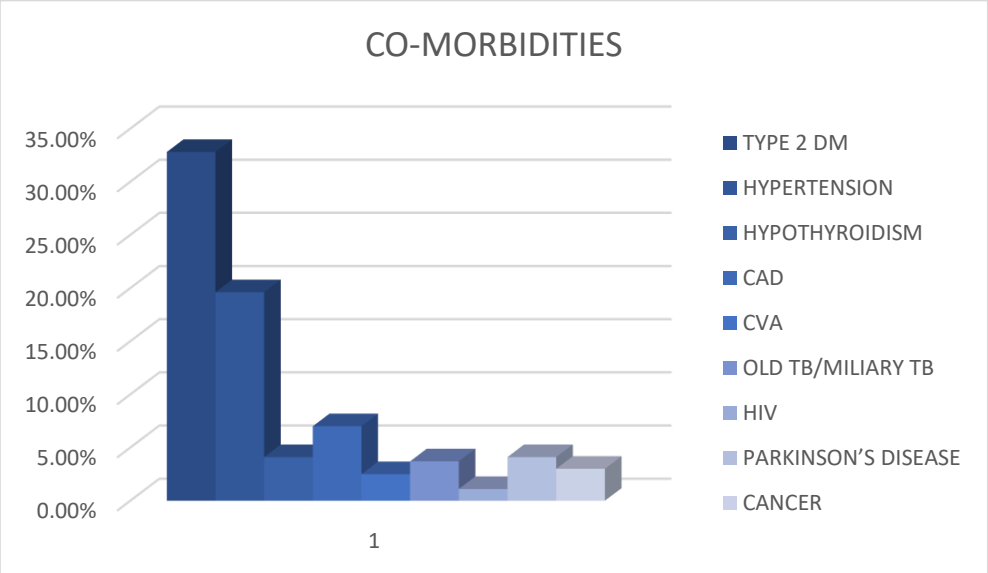
FIGURE 3: OTHER PRESNTING COMPLAINTS IN AFE

CO-MORBIDITIES:

Out of 271 patients presenting with Acute Febrile Encephalopathy, the most common co morbidity was TYPE 2 DM (32.8%).

TABLE 5: CO-MORBIDITIES IN STUDY GROUP PATIENTS (n-271)

CO-MORBIDITY	NO. OF PATIENTS	PERCENTAGE
TYPE 2 DM	89	32.8%
HYPERTENSION	53	19.6%
HYPOTHYROIDISM	11	4.1%
CAD	12	7.0%
CVA	7	2.5%
OLD TB/MILIARY TB	10	3.7%
HIV	3	1.1%
PARKINSON'S DISEASE	11	4.1%
CANCER	8	3.0%



FIGIRE 4: CO-MORBIDITIES IN PATIENTS WITH AFE

ETIOLOGICAL PROFILE

In our study it was found that Sepsis Associated Encephalopathy was the most common aetiology of acute febrile encephalopathy (36.5%) followed by TBM (29.2%) and Viral meningitis/ meningo-encephalitis (12.2%). Dengue associated Encephalopathy/ Encephalitis was found in 11.1% patients. Pyogenic Meningitis was found amongst 4.4% cases. 5 patients had Scrub related Encephalopathy, 4 presented with Cryptococcal Meningitis and 3 had Leptospira related aseptic meningitis, 3 were found to be Post Covid Encephalopathy after ruling out all other causes. 3 patients remained undiagnosed despite extensive imaging, all serologies and CSF analysis.

TABLE 6: ETIOLOGICAL PROFILE OF PATIENTS WITH AFE

DIAGNOSIS	No. of cases	Percentage
TBM	79	29.2%
Viral	33	12.2%
Dengue encephalopathy/ encephalitis	30	11.1%
Pyogenic	12	4.4%
Scrub related encephalopathy	5	1.8%
Sepsis associated encephalopathy	99	36.5%
Post covid encephalopathy	3	1.1%
Leptospira related encephalopathy	3	1.1%
Cryptococcal meningitis	4	1.5%
Undiagnosed	3	1.10%
Total	271	100%

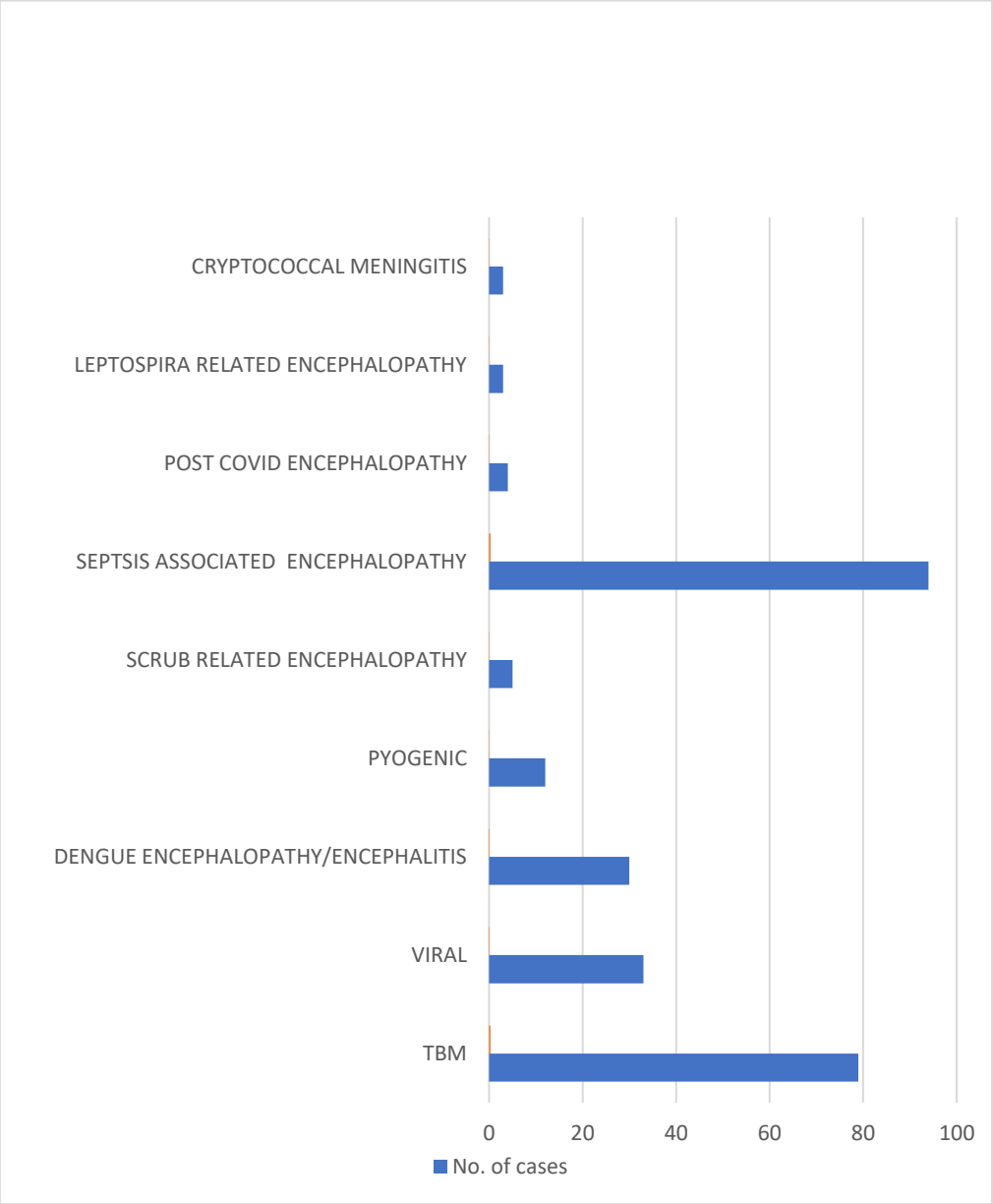


FIGURE 5: ETIOLOGICAL PROFILE OF PATIENTS HAVING AFE ENROLLED IN THE STUDY

RADIO-IMAGING:

CT HEAD:

In this table we can appreciate that in our study CT head was normal in most cases. Out of the cases where meningitis was present, the main CT head finding was hydrocephalus.

TABLE 7: CT HEAD FINDINGS IN PATIENTS OF AFE ENROLLED IN THE STUDY

CT HEAD FINDINGS	NO. OF CASES	PERCENTAGE
Focal hypodensities	11	4.1%
Hydrocephalus	23	8.4%
Diffuse cerebral oedema	5	1.8%
Infarct	3	0.11%
Others	8	2.9%
Normal	108	39.90%
Ct head not done	118	43.50%

CT HEAD FINDINGS:

1. Focal hypodensities were mainly found in patients with TBM (9 out of 11) and Viral Meningo-encephalitis (2 out of 11).
2. Hydrocephalus was found mostly in TBM (21 out of 23) and Pyogenic Meningitis (2 out of 23)
3. Diffuse Cerebral edema was mostly found in patients with Dengue Encephalopathy (4 out 5) and TBM (1 out of 5)
4. Infarct was demonstrated in 2 patients with TBM and one patient with SAE had a chronic infarct on his CT head.

5. Amongst significant other CT findings was multiple small haemorrhagic infarcts in a patient with Dengue Haemorrhagic Encephalitis.

MRI:

It was observed in our study that Leptomeningitis was the most common finding in patients presenting with Acute Febrile Encephalopathy.

TABLE 8: TABLE SHOWING MRI FINDINGS OF THE PATIENTS WITH ACUTE FEBRILE ENCEPHALOPATHY

MRI FINIDNGS	NO. OF PATIENTS	PERCENTAGE
Leptomeningitis	59	21.7%
Vasculitic infarct	33	12.1%
Ring enhancing lesions	27	9.9%
Meningoencephalitis/encephalitis	30	11.1%
Hydrocephalus	25	9.2%
Diffuse cerebral oedema	3	1.1%
Other findings	31	11.40%
MRI normal	45	16.60%
MRI not done	11	4.05%

MRI FINDINGS:

1. Out of the 59 patients who had Leptomeningitis evidence on MRI, 51 patients had Tuberculous Meningitis,6 patients had Pyogenic Meningitis and 2 patients had Cryptococcal Meningitis.

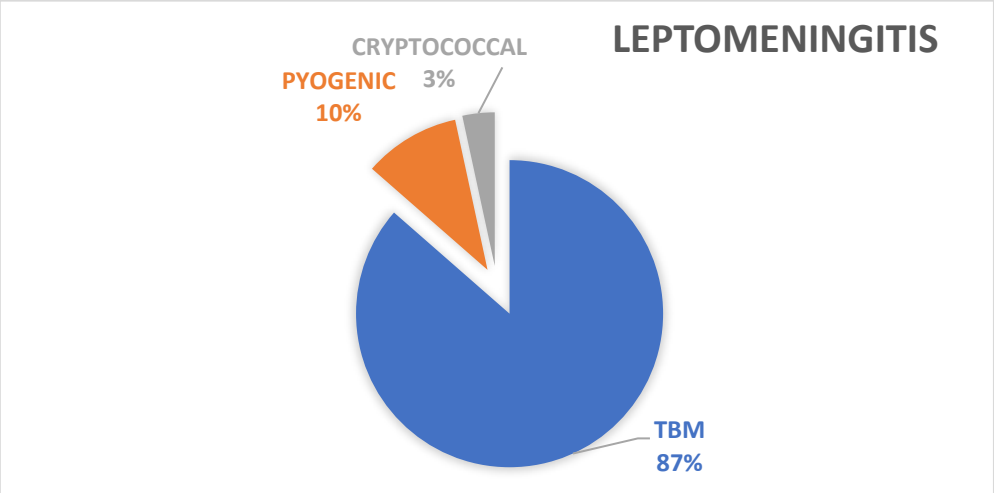


FIGURE 6: LEPTOMENINGITIS ON MRI BRAIN DIVIDED INTO ETIOLOGICAL ENTITIES

2. 34 patients out of 271 had Vasculitic Infarcts evident on MRI Brain, out of which 26 were TBM, 4 had Septic Emboli secondary to SAE,2 had Pyogenic Meningitis, 2 patient had Cryptococcal Meningitis.

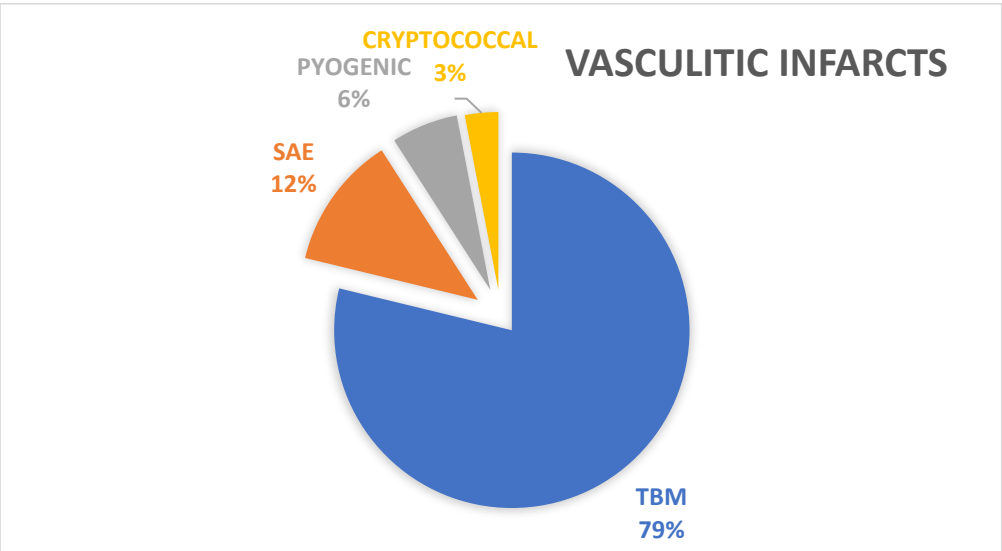


FIGURE 7: VASCULITIC INFARCTS ON MRI BRAIN DIVIDED INTO ETIOLOGICAL ENTITIES

3. 27 patients had evidence of ring enhancing lesions on MRI Brain and all were diagnosed with Tuberculous Meningitis.

4. 32 patients had e/o Encephalitis in their MRI Brain, 18 patients had Viral Encephalitis, 7 patients had TBM, 4 had Dengue Encephalitis, 2 Pyogenic Encephalitis and 1 had Scrub associated Encephalitis.

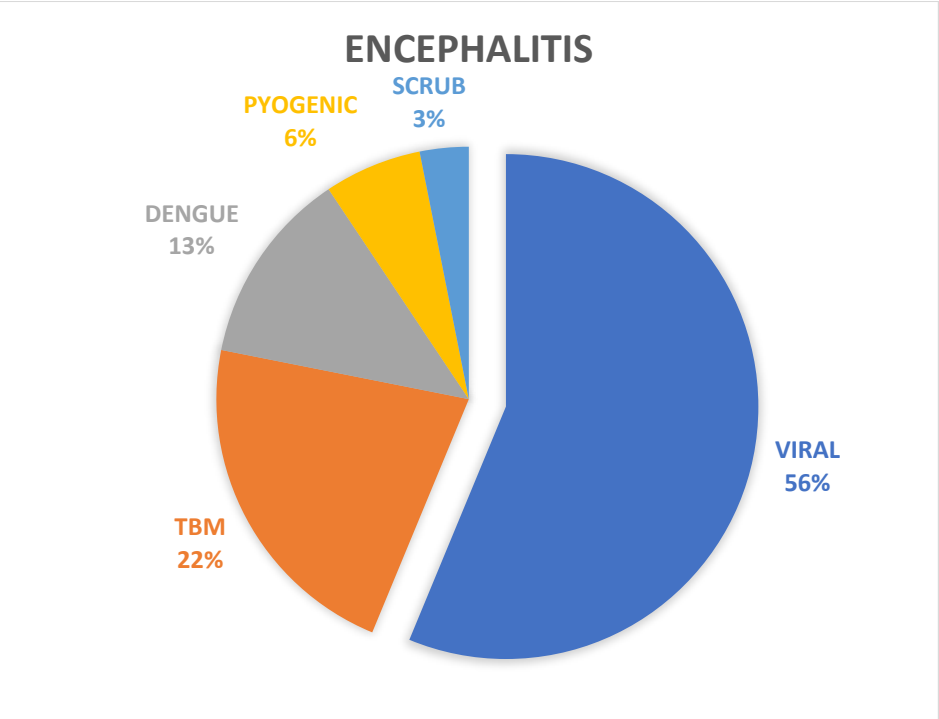


FIGURE 8: ENCEPHALITIS ON MRI BRAIN DIVIDED INTO ETIOLOGICAL ENTITIES

5. In 25 patients, Hydrocephalus was observed, 23 out of these were having TBM and 1 had Pyogenic Meningitis and 1 had chronic communicating Hydrocephalus but diagnosed with Sepsis Associated Encephalopathy.

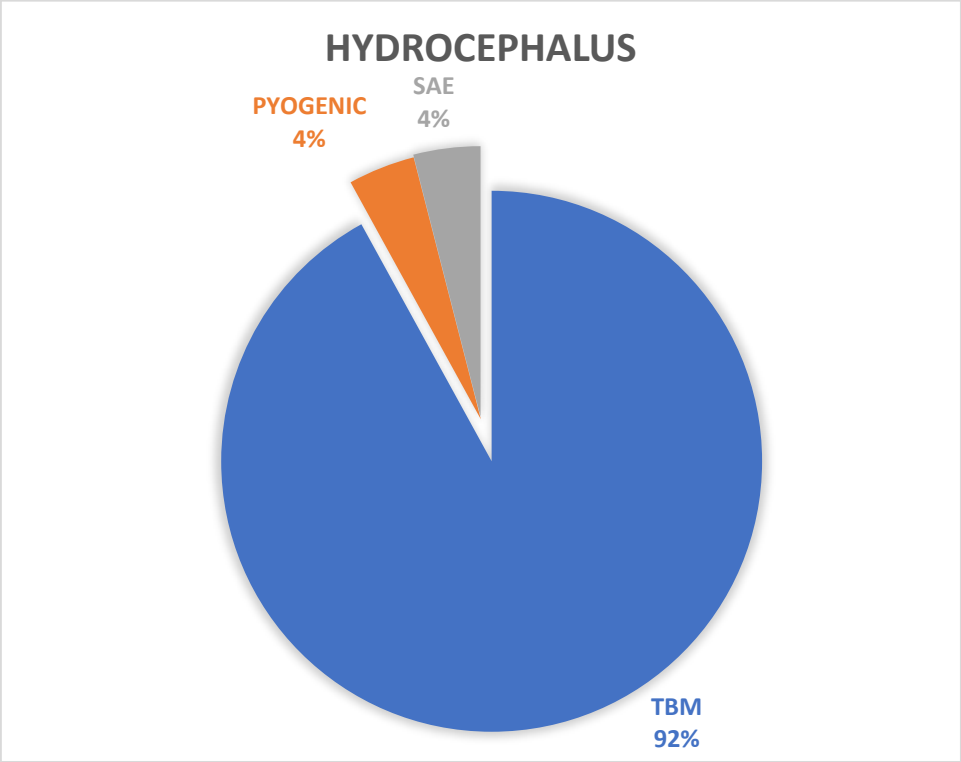


FIGURE 9: HYDROCEPHALUS ON MRI BRAIN DIVIDED INTO ETIOLOGICAL ENTITIES

6. Diffuse Cerebral Oedema was found in 3 patients, 1 with Dengue related Neurological complications, 1 with Viral Meningitis and 1 with Sepsis Associated Encephalopathy.
7. 1 patient with SAE had findings s/o PRES, 1 patient of Dengue Encephalitis had features of ADEM and 1 patient with Dengue Haemorrhagic manifestations had an ICH evident on MRI Brain
8. MRI was normal in 43 patients and not done in the rest.

MENINGITIS:

TUBERCULOUS MENINGITIS:

Out of 271 patients,79 patients were found to have Tuberculous Meningitis.

AGE DISTRIBUTION:

In our study, out of 79 total TBM patients, 34 belonged to 18 to 40 years age group, 30 belonged to 41to 60 years age group, more than 60 years patients were 15.

TABLE 9: AGE DISTRIBUTION IN TBM PATIENTS

AGE GROUP	NO. OF PATIENTS	PERCENTAGE
18 to 40 years	34	43%
41 to 60 years	30	38%
> 60 years	15	19%
Total	79	100%

SEX DISTRIBUTION:

In the present study, 42 out of 79 TBM patients were Male and rest were Female. Sex ratio was 0.88.

TABLE 10: SEX DISTRIBUTION IN TBM PATIENTS

SEX	NO. OF PATIENTS	PERCENTAGE
Male	42	53%
Female	37	47%
Total	79	100%

PRESENTING COMPLAINTS:

Apart from fever and altered sensorium which was found in all TBM patients, most common chief complaint was Headache

TABLE 11: CHIEF COMPLAINTS OF PATIENTS WITH TBM

PRESENTING COMPLAINTS	NO. OF PATIENTS
Headache	33
Vomiting	19
Abnormal body movements	11
Diplopia	10
Weakness in any part of body	6

SIGNIFICANT CO-RELATIONS:

Cartridge based nucleic acid amplification (CBNAAT) detects DNA sequences specific for Mycobacterium tuberculosis and Rifampicin resistance by PCR. It has High sensitivity and specificity. Since Biopsy is an invasive procedure and not warranted in patients where other tests are available to make reliable diagnosis and AFB C/S was not sent in all patients, all CBNAAT positive cases are considered to have Tuberculous Meningitis in this study.

TABLE 12: TABLE SHOWING CBNAAT IN CSF POSITIVE CASES AMONGST TOTAL CASES OF TUBERCULOUS MENINGITIS.

CBNAAT	NO. OF TBM CASES	PERCENTAGE
Negative	53	65.8%
Positive	26	34.2%
Total	79	100.0%

Amongst the 79 patients who were diagnosed with TBM, 26 tested positive for CBNAAT in CSF.

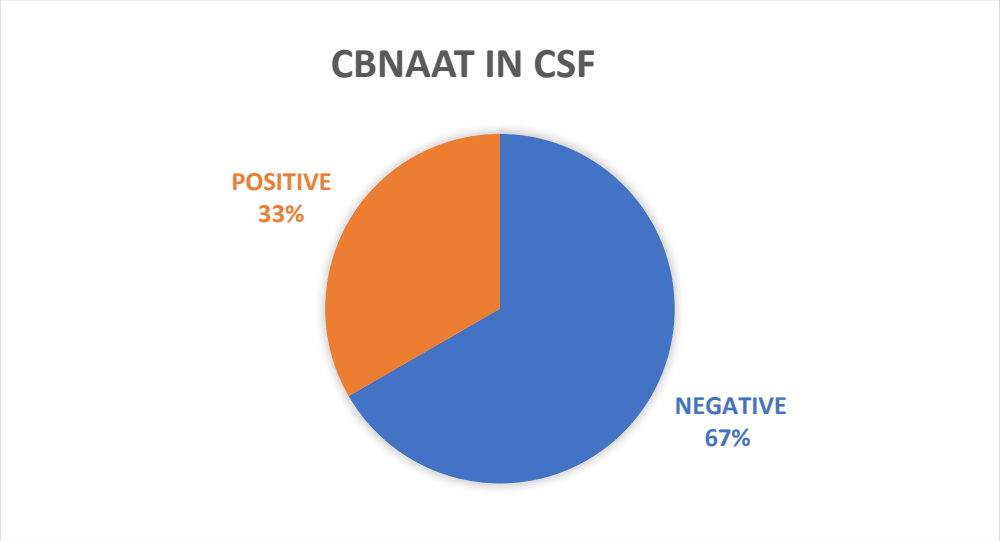


FIGURE 10: POSITIVE CBNAAT IN CSF CASES OUT OF TOTAL TBM PATIENTS.

CSF FINDINGS IN TUBERCULOUS MENINGITIS:

Tuberculous Meningitis was diagnosed on the basis of CSF findings including significant TLC count, lymphocytic predominance, low CSF glucose, high CSF protein levels and High CSF ADA levels. Positive CBNAAT in CSF also confirmed TBM but was not present in all patients. MRI findings supported the diagnosis and some MRI findings like Hydrocephalus also determined prognosis and outcome.

TABLE 13: CSF ADA LEVELS IN PATIENTS WITH TBM

ADA LEVELS	NO. OF PATIENTS
ADA > 10 IU/L	63
ADA <10 IU/L	9
ADA NOT DONE	7
TOTAL	79

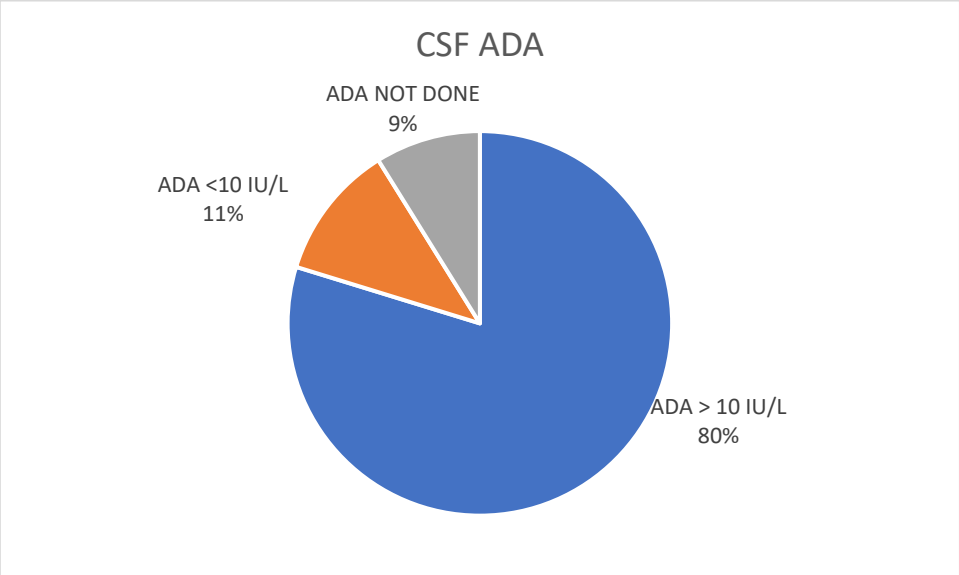


FIGURE 11: CSF ADA LEVELS IN PATIENTS WITH TBM

CSF PROTEIN LEVELS:

TABLE 14: CSF PROTEIN LEVELS IN PATIENTS WITH TBM(n-79)

PROTEIN LEVELS	NO. OF PATIENTS
< 60 mg/dL	7
>60 mg/dL	71
NOT DONE	1
TOTAL	79

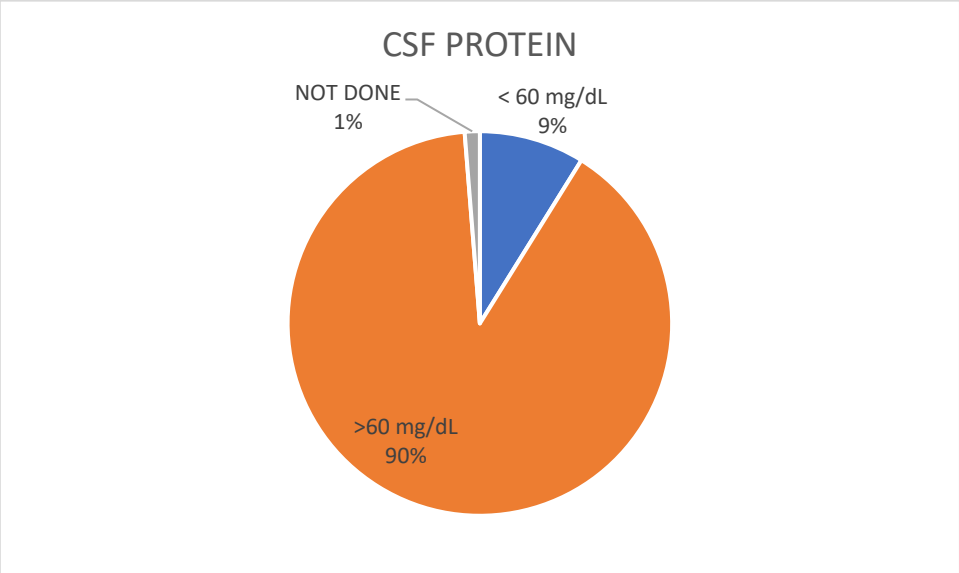


FIGURE 12: CSF PROTEIN LEVELS IN PATIENTS WITH TBM

CSF GLUCOSE LEVELS:

56 patients out of 79 had CSF: serum glucose ratio <0.5.

TABLE 15: CSF: SERUM GLUCOSE <0.5 IN TBM (Total-79)

CSF: SERUM GLUCOSE	NO. OF PATIENTS
<0.5	56
>0.5	23
NOT DONE	1

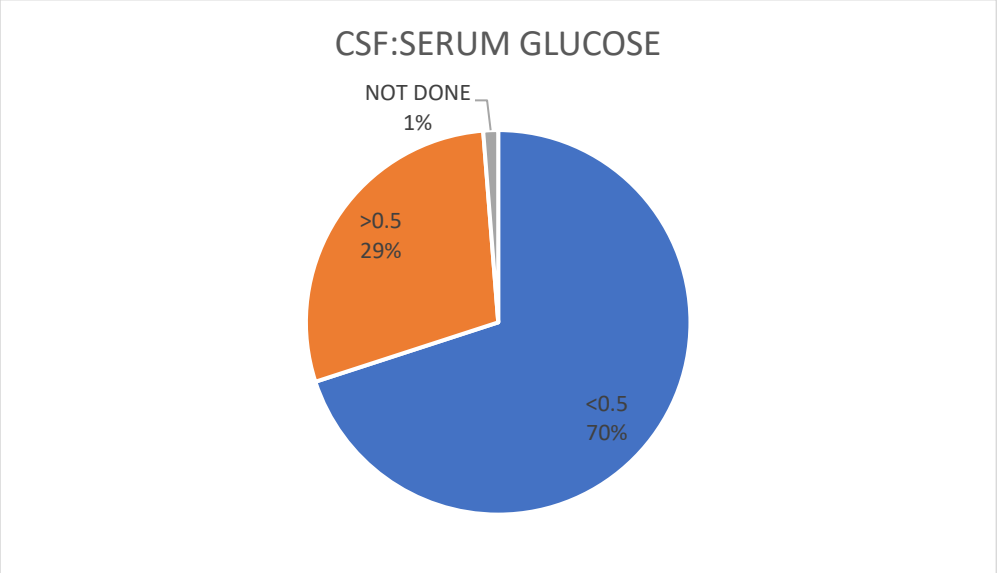


FIGURE 13: CSF: SERUM GLUCOSE <0.5 IN TBM PATIENTS

Amongst the patients who tested ADA negative, all of them had low CSF glucose and high CSF protein levels except one where CSF analysis was not possible. They were still diagnosed with TBM as three of them had CSF CBNAAT positivity, 2 had characteristic Tuberculomas in MRI Brain, 2 had Leptomeningitis and vasculitic infarcts and one only had Leptomeningitis.

Amongst the patients in whom CSF ADA had not been sent, 5 out of 7 patients had CBNAAT positivity. 1 had characteristic MRI findings of Leptomeningitis, vascular infarct and tuberculoma and 1 was diagnosed with TBM on basis of clinical features, CSF findings of predominant lymphocytosis, low glucose and high protein levels.

SENSTIVITY OF CSF ADA IN DIAGNOSING TBM

Out of 26 patients who were CBNAAT positive in CSF, 19 had significant ADA, 3 had ADA< 10 IU/Dl and 4 did not get their CSF ADA done, hence the sensitivity of CSF ADA in diagnosing TBM came out to be 90.4%.

TABLE 16: SENSITIVITY OF CSF ADA IN DIAGNOSING TBM

CSF ADA	CBNAAT NEGATIVE	CBNAAT POSITIVE
<10 U/L	6	3
>10 U/L	45	19
NOT DONE	2	4
TOTAL	53	26

SENSITIVITY OF CSF GLUCOSE IN DIAGNOSING TBM:

In patients who had CBNAAT positivity (26), 24 had a significantly low CSF glucose, hence the sensitivity of CSF Glucose in diagnosing TBM was 92.3%.

TABLE 17: SENSITIVITY OF CSF GLUCOSE IN DIAGNOSING TBM

CSF:SERUM GLUCOSE	CBNAAT NEGATIVE	CBNAAT POSITIVE
<0.5	45	24
>0.5	7	2
NOT DONE	1	0
TOTAL	53	26

SENSITIVITY OF CSF PROTEIN:

Out of 26 patients who had CBNAAT positivity in their CSF, all of them had significantly high CSF protein values. Hence the sensitivity of this test is 100%.

TABLE 18: SENSITIVITY OF CSF PROTEIN IN DIAGNOSING TBM

CSF PROTEIN	CBNAAT NEGATIVE	CBNAAT POSITIVE
>60 mg/dL	45	26
<60 mg/dL	7	0
NOT DONE	1	0
TOTAL	53	26

RADIOIMAGING FINDINGS:

Out of 26 patients diagnosed with TBM on the basis of CBNAAT positivity in CSF,11 patients had drowsiness/seizures/GCS<10 or weakness, out of these 11 patients, the most common MRI finding was found to be Hydrocephalus found in 7 patients,5 had ring enhancing lesions s/o tuberculomas, 4 had vasculitic infarcts. Leptomeningitis was found in combination with the other three findings mentioned above in 8 out of 11 patients and only 1 patient with low GCS had Leptomeningitis alone.1 patient had meningoencephalitis also.

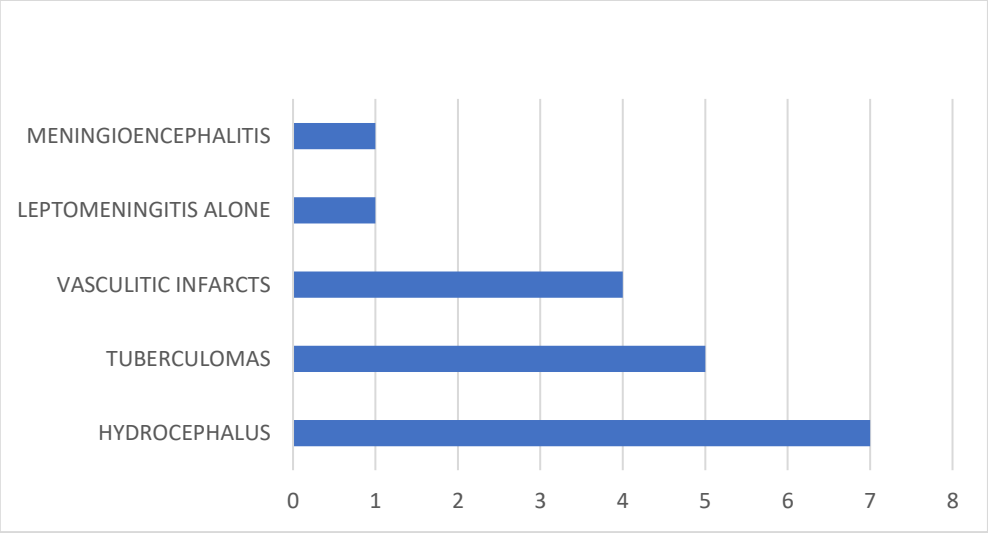


FIGURE 14: MRI FINDINGS IN PATIENTS WITH CBNAAT POSITIVE TBM WITH LOW GCS (<10)

PYOGENIC MENINGITIS:

In our study 12 out of 271 patients were diagnosed with Pyogenic Meningitis.

AGE DISTRIBUTION:

No age group predominance found in this group.

TABLE 19: AGE DISTRIBUTION IN PYOGENIC MENINGITIS

(Total -12)

AGE GROUP	NO. OF PATIENTS	PERCENTAGE
18-40 YEARS	4	33%
40-60 YEARS	3	25%
>60 YEARS	5	42%

SEX DISTRIBUTION:

In this group the male to female ratio was 1:1

TABLE 20: SEX DISTRIBUTION IN PYOGENIC MENINGITIS

(Total-12)

SEX DISTRIBUTION	NO. OF PATIENTS	PERCENTAGE
MALE	6	50%
FEMALE	6	50%

CSF FINDINGS:

In this group of patients, Latex Antigen was found to be positive in 3 patients for *Streptococcus pneumoniae*. 2 CSF cultures came out to be positive, one for S. typhi and second for *Klebsiella pneumoniae*.

TABLE 21: CSF FINDINGS IN PYOGENIC MENINGITIS

CSF FINDINGS	NO. OF PATIENTS
LATEX ANTIGEN POSITIVE	3
CSF C/S POSITIVE	2
NEUTROPHIL PREDOMINANCE WITH HIGH CSF PROTEIN AND	7
TOTAL	12

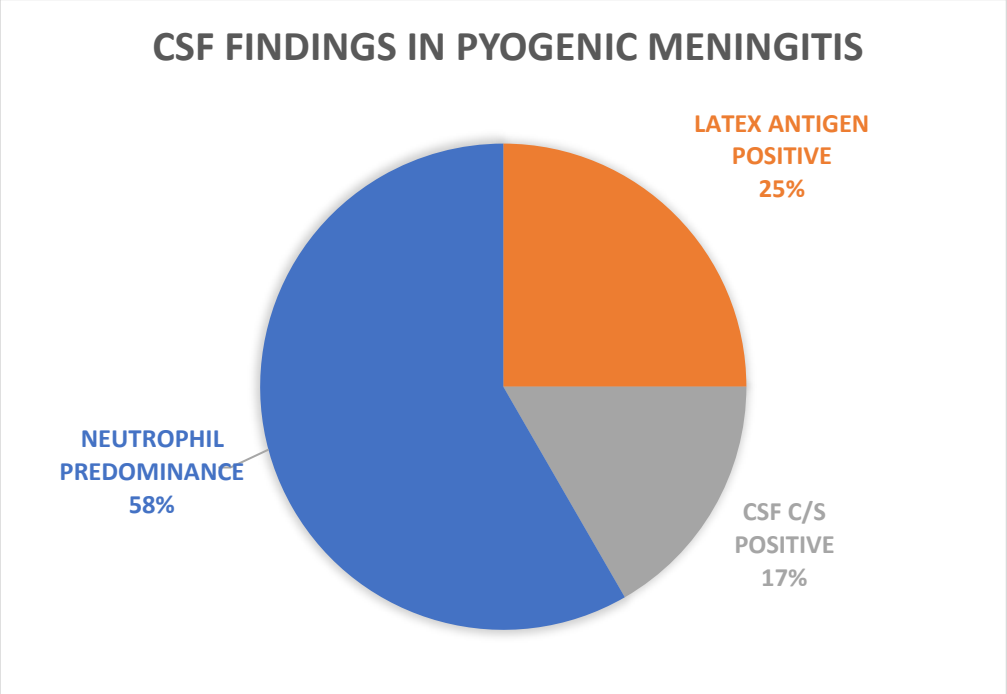


FIGURE 15: CSF FINDINGS IN PYOGENIC MENINGITIS

IMAGING FINDINGS:

In Pyogenic Meningitis, MRI was s/o Leptomeningitis in 6 patients, vasculitic infarct in 2 patients, Meningo encephalitis in 2 patients, 1 patient had hydrocephalus and in 1 patient MRI was normal.

TABLE 22: MRI FINDINGS IN PYOGENIC MENINGITIS

MRI FINDINGS	NO. OF PATIENTS
LEPTOMENINGITIS	6
VASCULITIC INFARCTS	2
MENINGO ENCEPHALITIS	2
HYDROCEPHALUS	1
NORMAL	1
TOTAL	12

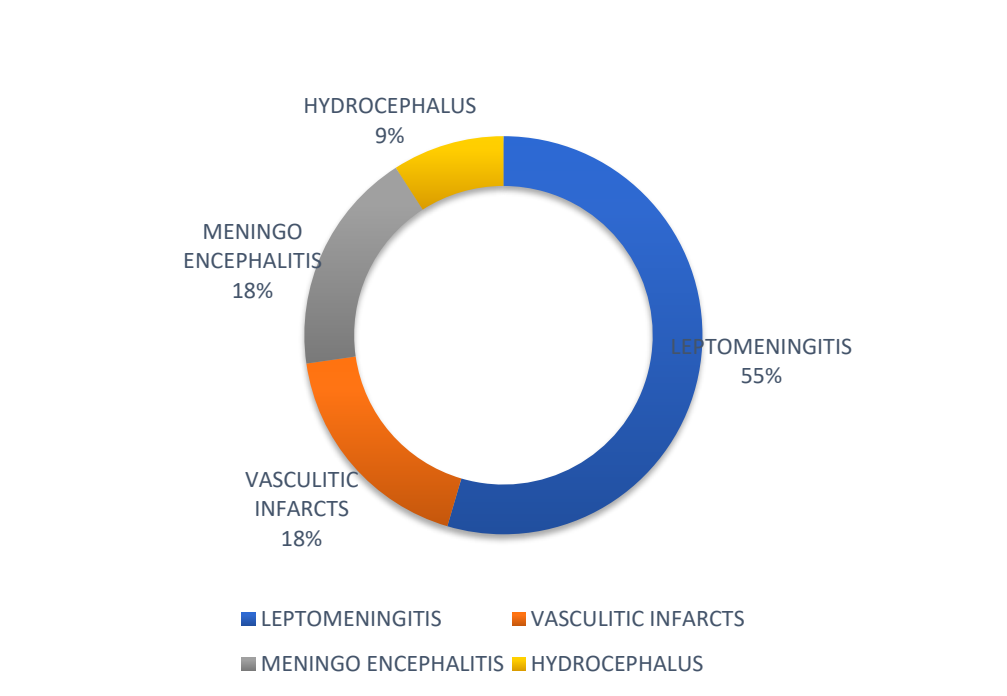


FIGURE 16: IMAGING FINDINGS IN PATIENTS WITH PYOGENIC MENINGITIS.

VIRAL MENINGITIS/MENINGO ENCEPHALITIS:

AGE DISTRIBUTION:

In our study, 33 patients were diagnosed with Viral Meningitis/Meningo encephalitis. Following table demonstrates their age distribution.

TABLE 23: AGE DISTRIBUTION IN VIRAL MENINGO-ENCEPHALITIS

AGE GROUPS	NO. OF PATIENTS
18 TO 40 years	11
41 to 60years	13
>60 years	9
TOTAL	33

SEX DISTRIBUTION

Patients with Viral Meningo encephalitis had a male: female ratio of 1.53:1.

TABLE 24: SEX DISTRIBUTION IN VIRAL MENINGO ENCEPHALITIS

SEX DISTRIBUTION	NO. OF PATIENTS
MALE	20
FEMALE	13
TOTAL	33

PRESNTING COMPLAINTS:

Among patients with Viral Meningo-encephalitis, abnormal body movements were the most common presenting complaint after fever and altered sensorium.

TABLE 25: PRESENTING COMPLAINTS IN VIRAL MENINGO-ENCEPHALITIS

PRESENTING COMPLAINTS	NO. OF PATIENTS
Abnormal body movements	19
Headache	8
Vomiting	6
Total	33

SIGNIFICANT CO-RELATIONS:

Out of 33 total cases diagnosed with Viral meningitis/ meningo-encephalitis, 16 patient's CSF sample were HSV PCR positive.

TABLE 26: HSV PCR POSITIVE CASES AMONGST TOTAL VIRAL MENINGO-ENCEPHALITIS PATIENTS

HSV PCR IN CSF	NO. OF PATIENTS
HSV PCR POSITIVE	16
HSV PCR NEGATIVE/NOT SENT	17
TOTAL	33

It was found that out of 16 patients, 11 patients with HSV PCR positivity in CSF had MRI finding s/o encephalitis. The 5 patients who did not have e/o meningeal enhancement or encephalitis on MRI Brain, one did not get a neuroimaging done, 2 patients had a normal MRI brain and one had transient splenial lesion and one had cystic encephalomalacia. The patients with Normal MRI Brain were likely the cases of Aseptic meningitis due to viral etiology.

TABLE 27: VIRAL MENINGO-ENCEPHALITIS PATIENTS WITH CSF HSV PCR POSITIVE (TOTAL = 16)

HSV PCR POSITIVE IN CSF AND MRI CO-RELATION	NO. OF PATIENTS
HSV POSITIVE WITH MRI S/O ENCEPHALITIS	11
HSV POSITIVE WITH MRI NOT S/O ENCEPHALITIS	5

The patients in which HSV PCR was not found in the CSF but were still classified as Viral Meningitis/meningo-encephalitis were 17 in number. Out of them 6 patients had MRI findings specific for Herpes Encephalitis and 11 were put into this category because of CSF findings (commonly, normal glucose, higher protein levels with low ADA levels, significant TLC in CSF was found only in 3 out of 11 of these patients) and clinical picture.

TABLE 28: VIRAL MENINGITIS/ENCEPHALITIS PATIENTS WITH NEGATIVE HSV PCR (TOTAL = 17)

HSV PCR NEGATIVE PATIENTS AND MRI CO-RELATION	NO. OF PATIENTS
HSV NEGATIVE WITH MRI S/O HERPES ENCEPHALITIS	6
HSV NEGATIVE WITH NO SPECIFIC MRI FINDING	11

SEPSIS ASSOCIATED ENCEPHALOPATHY:

In the present study 99 out of 271 patients were diagnosed as Sepsis Associated Encephalopathy. A pre requisition for diagnosis of SAE is presence of Sepsis and presence of features of encephalopathy.

Sepsis Associated Encephalopathy is a diagnosis of exclusion. Only those patients were labelled with this diagnosis in the present study who got at least one neuroimaging done to rule out CVA and structural causes. CSF if done was not s/o CNS infection in this group though mild rise in CSF protein was found in some cases. Also, in all the patients there was evidence of sepsis either clinically in form of history of high-grade fever, high TLC, Leucopenia or evidence of septicemia in form of growth in Blood c/s or raised Procalcitonin or source of sepsis identified on other cultures or radio-imaging. In some patients MRI Brain was s/o non-specific findings related to SAE.

AGE DISTRIBUTION:

In this group, the patients who were aged > 60 years were significantly higher than the other groups. 68 out of 99 patients of SAE were > 60 years of age.

TABLE 29: AGE DISTRIBUTION OF PATIENTS WITH SAE

AGE GROUPS	NO. OF PATIENTS
18 - 40 YEARS	10
41 - 60 YEARS	21
> 60 YEARS	68
TOTAL	99

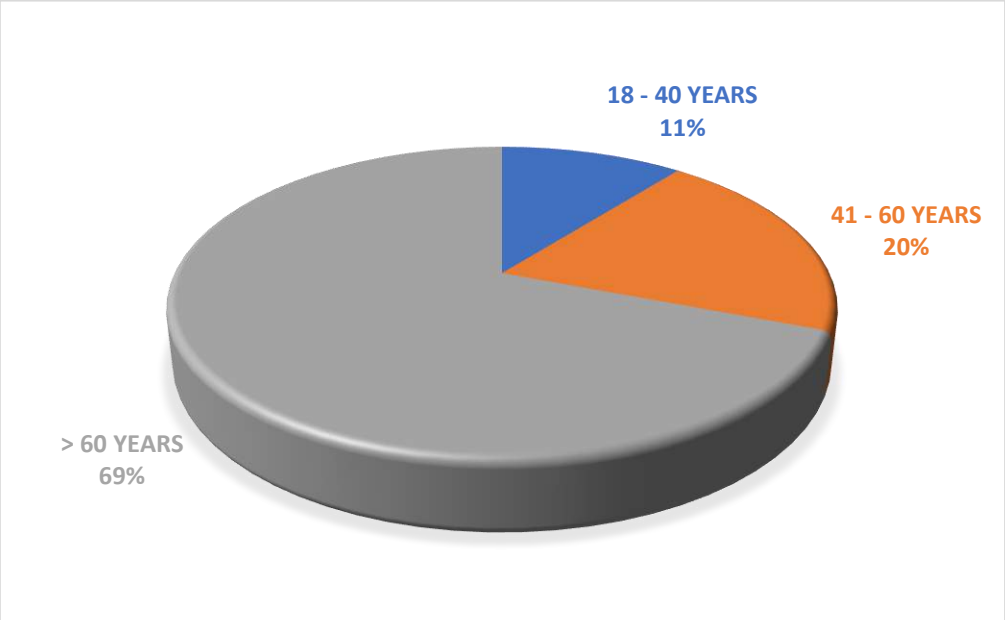


FIGURE 17: AGE DISTRIBUTION IN PATIENTS WITH SAE

SEX DISTRIBUTION:

In our study we observed that out of 99 patients who were diagnosed with SAE, 67 were males and the sex ratio was 2.09, hence males were significantly more affected than females in this group.

TABLE 30: SEX DISTRIBUTION OF PATIENTS WITH SAE

SEX DISTRIBUTION	NO. OF PATIENTS
MALE	67
FEMALE	32
TOTAL	99

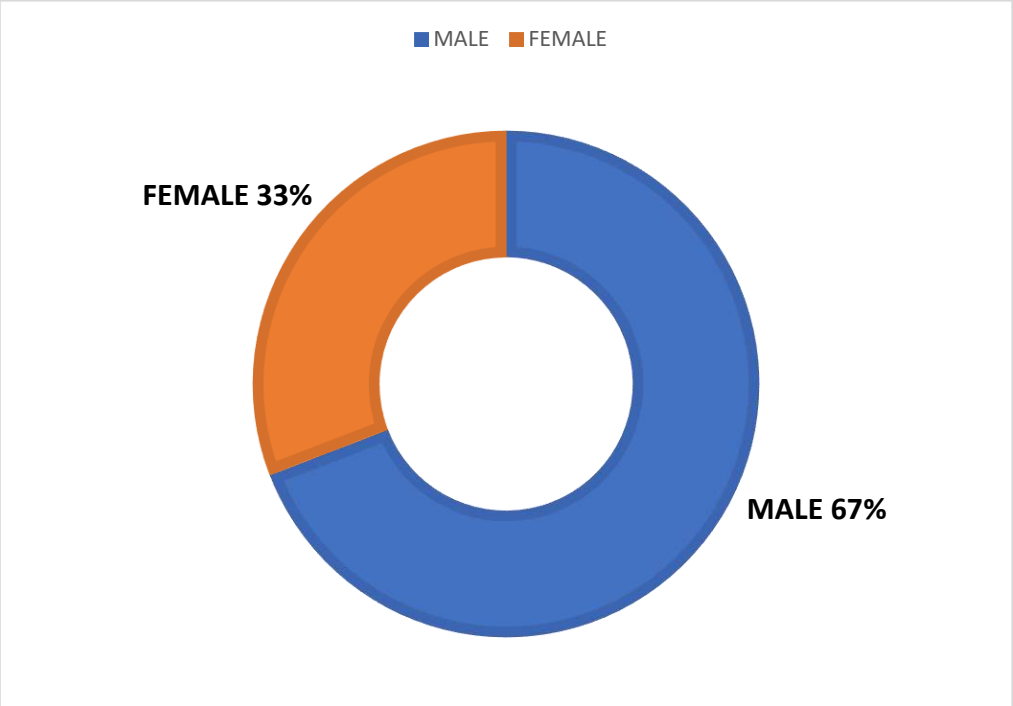


FIGURE 18: SEX DISTRIBUTION OF PATIENTS WITH SAE

PRESENTING COMPLAINTS

It was found that patients in this group had a wide heterogeneity in their chief complaints which brought them to seek healthcare. 11 out 99 patients had headache and seizures occurred in 9 patients with SAE, other symptoms were decreased urine output, pyuria, shortness of breath, cough, abdominal pain, loose stools, foot or leg swellings, bed sores etc.

CO-MORBIDITIES:

In Sepsis Associated Encephalopathy, TYPE 2 DM was the most common co-morbidity. 53 out of 99 patients TYPE 2 DM,23 had Hypertension.

TABLE 31: CO-MORBIDITIES IN PATIENTS WITH SAE

CO-MORBIDITIES	NO. OF PATIENTS
Type 2 dm	53
Hypertension	23
CAD/CHF	10
COAD	15
Malignant disease	6
No co morbidities	32

CULTURE SENSITIVITY:

It was found that out of 271 patients, 75 were found to have growth of an organism from various samples taken for culture sensitivity.

TABLE 32: POSITIVE C/S OUT OF TOTAL STUDY POPULATION

C/s POSITIVE	NO OF CASES	PERCENTAGE
YES	75	27%
NO	196	73%
TOTAL	271	100%

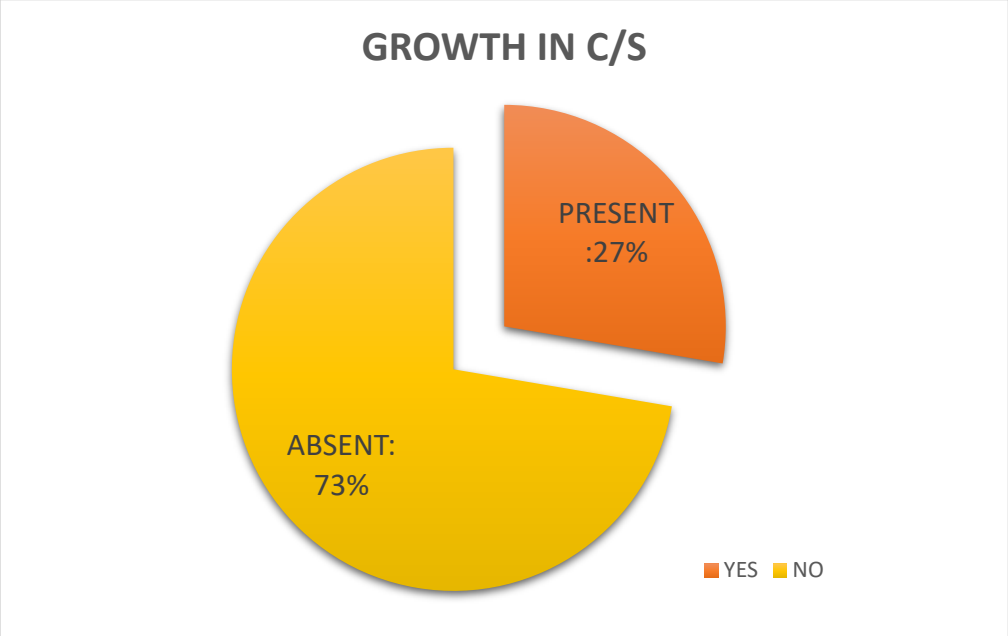


FIGURE 19: POSITIVE C/S OUT OF TOTAL STUDY POPULATION

Among total 75 patients who had a positive culture growth, 59 were diagnosed to have Sepsis Associated Encephalopathy

TABLE 33: C/S POSITIVITY IN SAE PATIENTS

PATIENTS WITH SAE	NO. OF CASES	%AGE
SAE WITH C/S POSITIVE	59	59.5%
SAE WITH NO GROWTH ON C/S	40	40.5%
TOTAL	99	100%

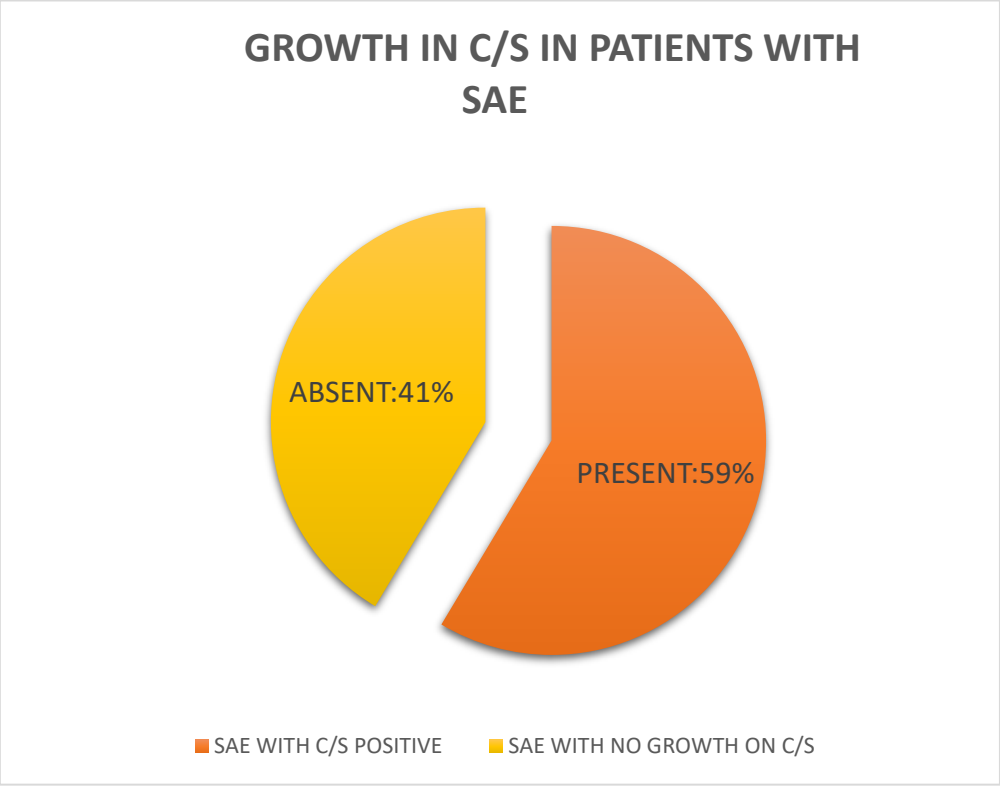


FIGURE 20: C/S POSITIVITY IN SAE PATIENTS

Among patients with positive C/S, total 64 sites of infection were found. Out of these 64, 5 patients had double site infection, and 54 had single site infection.

TABLE 34: TOTAL NUMBER OF INFECTION SITES IN PATIENTS WITH POSITIVE C/S IN SAE

NO. OF INFECTION SITES	NO. OF PATIENTS
Single site infection	54
Double site infection	5
Total no. of infections	64

Out of the total 64 infection sites or sources that were observed in our study in patients with SAE, the most common source/site was UTI followed by Septicemia, Respiratory Infections and then skin and soft tissue infections.

TABLE 35: SOURCES OF ISOLATES OBTAINED IN C/S OF VARIOUS SAMPLES IN PATIENTS WITH SAE

SOURCE OF ISOLATE (TOTAL INFECTIONS)	NO. OF PATIENTS	%Age
UTI	32	50%
RTI	11	17%
Bacteremia	12	19%
SSTI	9	14%
Total infections	64	100%

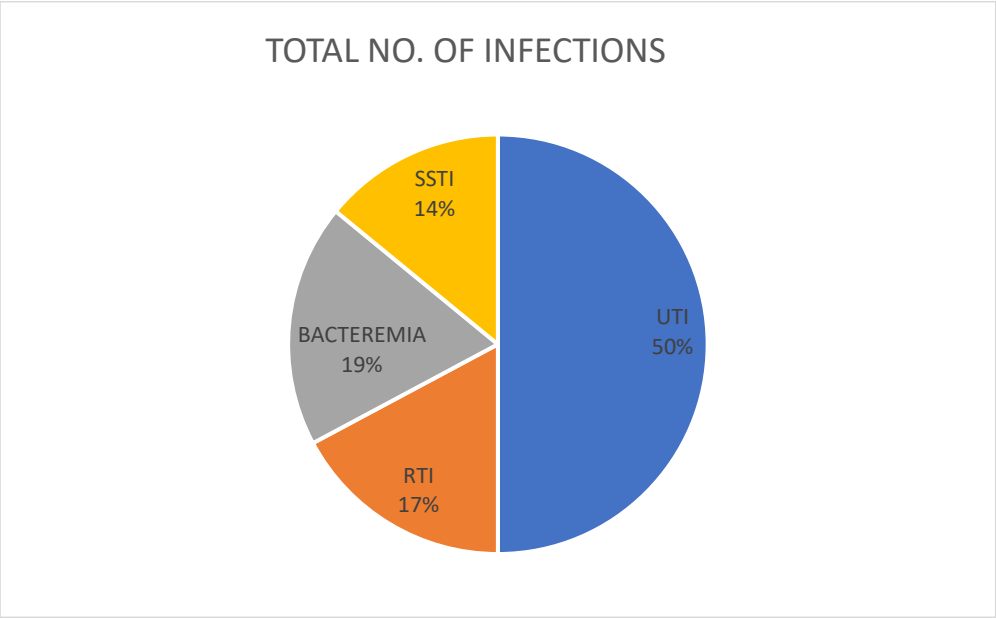


FIGURE 21: SOURCES OF ISOLATES OBTAINED IN C/S OF VARIOUS SAMPLES IN PATIENTS WITH SAE

Out of the total 64 infections, the most common organism isolated was E.coli followed by Klebsiella, Staphylococcus aureus and Pseudomonas. The most common source of infection was found to be UTI.

TABLE 36: DISCRIPTION OF ALL THE ISOLATES OBTAINED IN PATIENTS WITH SAE.

	SEPTICEMIA	UTI	RTI	SSTI	TOTAL
E.coli	1	18	0	2	21
Klebsiella	2	6	7	1	16
Pseudomonas/ burkholderia	3	2	3	1	9
Proteus		1			1
Enterococcus	1	3	0	1	5
Staphylococcus aureus	3	1		4	8
Aspergillus	1				1
Elizabethkingia	1				1
Candida		1			1
Acinetobacter			1		1
Total	12	32	11	9	64

Apart from proven C/S positivity, many patients presented with symptoms involving a particular system along with features of sepsis. Though there was no C/S growth obtained when abdominal source was considered to be the source of sepsis but in view of presenting symptoms like abdominal pain, loose motions, high grade fever, vomiting, jaundice etc. along with features of SAE and imaging findings showing source of sepsis. Eg. Liver Abscess, cholangitis etc., source of infection was documented to be Abdomen. Similarly imaging evidence for Septic arthritis, pyometra, Pyelitis, Emphysematous cystitis etc helped in delineating source of sepsis. Out of 99, the source of sepsis of 85 patients could be established based on either the C/S positivity or the presenting symptoms and imaging findings as mentioned above. The most common source in our study was found to be Urosepsis followed by Respiratory tract infection, Bacteremia and abdominal infections.

TABLE 37: SOURCE OF SEPSIS IN SAE PATIENTS

SOURCE OF SEPSIS	NO. OF PATIENTS	% AGE
UTI	35	36%
RTI	15	15%
BACTEREMIA	13	13%
SSTI	10	10%
ABDOMINAL	12	12%
UN-IDENTIFIED	14	14%
TOTAL	99	100%

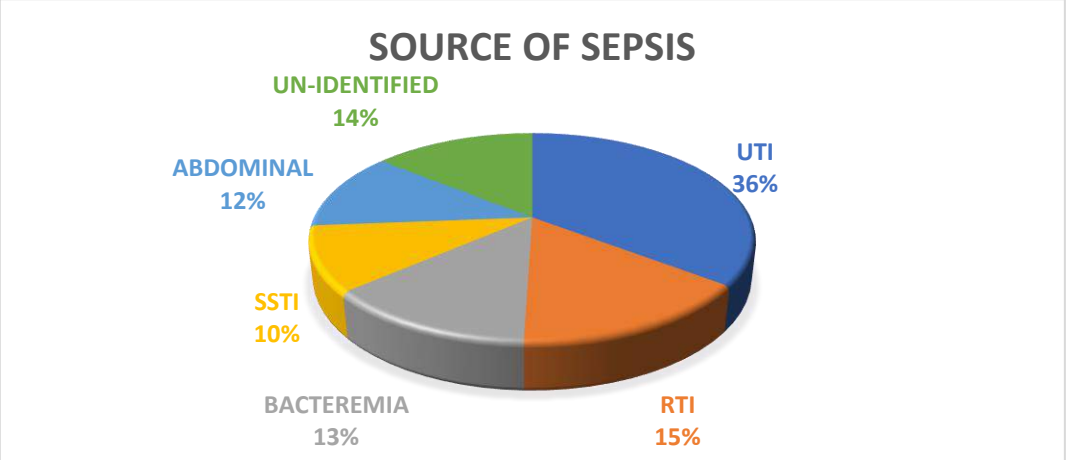


FIGURE 22: SOURCES OF SEPSIS IN PATIENTS WITH SAE BASED ON C/S POSITIVITY AND CLINICAL PRESENTATION.

PROCALCITONIN:

In this study, the patients with Sepsis Associated Encephalopathy were 99 in number. Out of these PCT was done in 86 patients.

1. Patients with SAE and Significant PCT values (>0.05 ng/dL): 64
2. Patients with SAE and Insignificant PCT value (<0.05 ng/dL): 22
3. Patients with SAE with positive c/s and significant PCT: 42
4. Patients with SAE without positive organism growth in body fluid c/s with significant PCT: 22
5. Patients with SAE without positive organism growth in body fluid c/s with insignificant PCT: 13

The 13 patients where neither there was c/s positivity in body fluids, nor the PCT was high were diagnosed with SAE on basis of negative neuroimaging/CSF findings, positive fungal markers or Imaging findings s/o septic foci along with clinical features of sepsis like high grade fever, high TLC, Leukopenia, tachycardia etc.

TABLE 38: SIGNIFICANCE OF PCT IN SAE (n-86)

SAE AND PCT CORELATION	C/S GROWTH PRESENT	NO C/S GROWTH	TOTAL
SAE WITH RAISED PCT	42	22	64
SAE WITHOUT RAISED PCT	9	13	22
TOTAL	51	35	86

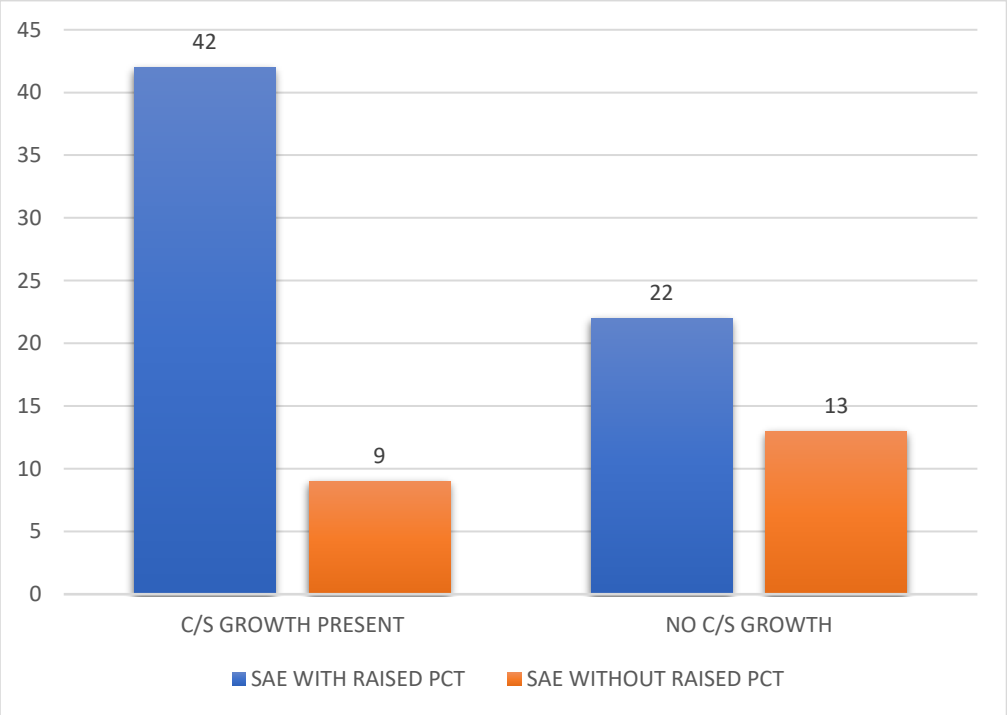


FIGURE 23: SIGNIFICANCE OF PCT IN SAE

SIGNIFICANT RADIO-IMAGING FINDINGS:

4 patients had e/o acute infarcts, 2 MRI pictures were s/o cytotoxic oedema, sub cortical white matter hyperintensities found in 1 patient, leukoencephalopathy evident in 1 patient,1 MRI was also s/o chronic hydrocephalus and diffuse cerebral oedema found in 1 patient.1 patient also had features of PRES.

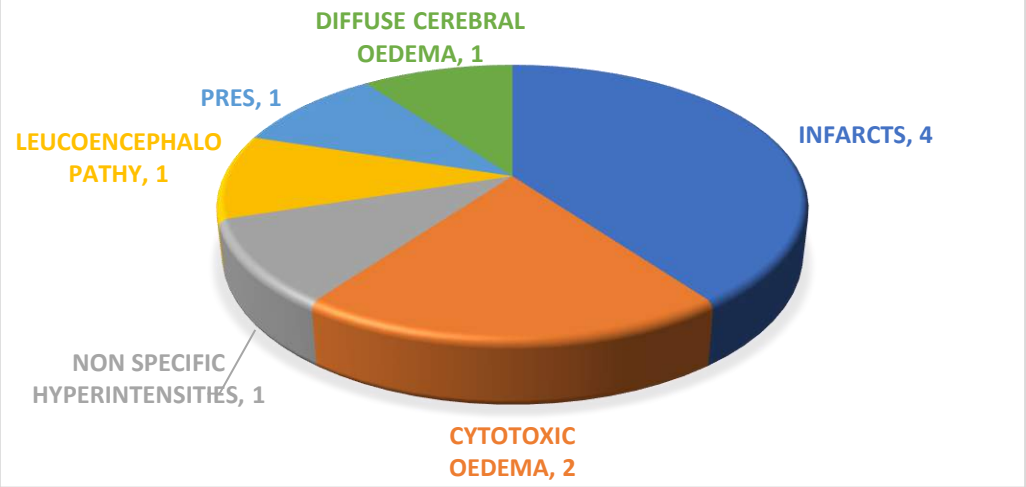


FIGURE 24: MRI FINDINGS OF PATIENTS WITH SAE67

SAE AND HYPOTENSION:

In our study, out of 99 patients with SAE, 57 (57.7%) were in shock on presentation

TABLE 39: HYPOTENSION IN PATIENTS WITH SAE

SEPTSIS ASSOCIATED ENCEPHALOPATHY		
HYPOTENSION PRESENT	57	57.57%
HYPOTENSION ABSENT	42	42.42%
TOTAL	99	100%

Also, when compared to patients with organism growth in their body fluids, 27 out of 58 of these patients presented with septic shock. The outcome of these findings has been discussed later.

TABLE 40: HYPOTENSION IN PATIENTS WITH CULTURE POSITIVE SAE

SAE WITH POSITIVE CULTURE GROWTH (TOTAL-58)		
HYPOTENSION PRESENT	27	46.60%
HYPOTENSION ABSENT	31	53.40%

DENGUE ENCEPHALOPATHY/ENCEPHALITIS:

In this study 30 patients out of 271 were diagnosed with Dengue Encephalopathy/ Encephalitis.

AGE DISTRIBUTION:

In this group of patients, maximum patients were found in the age group of 40 to 60 years

TABLE 41: AGE DISTRIBUTION OF PATIENTS WITH DENGUE ASSOCIATED NEUROLOGICAL COMPLICATIONS

AGE GROUP	NO. OF PATIENTS (TOTAL-30)
18-40 YEARS	8
40-60 YEARS	14
>60 YEARS	8

SEX DISTRIBUTION:

Among 30 patients in this group, 17 were male and rest females.

TABLE 42: SEX DISTRIBUTION OF PATIENTS WITH DENGUE ASSOCIATED NEUROLOGICAL COMPLICATIONS

SEX DISTRIBUTION	NO. OF PATIENTS (TOTAL-30)
MALE	17
FEMALE	13

SIGNIFICANT CO-RELATIONS:

RAISED AMINOTRANSFERASE LEVELS

As demonstrated in previous studies also, the patients having Dengue related neurological complications were found to have significantly raised hepatic transaminases. In this study, 22 out of 30 patients who were found to have Dengue related neurological complications were found to have raised hepatic transaminases, SGOT > SGPT, at least 5 times above the normal limit.

TABLE 43: RAISED HEPATIC TRANSAMINASES IN PATIENTS WITH DENGUE ENCEPHLOPATHY

RAISED AMINOTRANSFERASES IN DENGUE ENCEPHALOPATHY		**%AGE**
YES	22	73.3%
NO	8	26.6%
TOTAL	30	100%

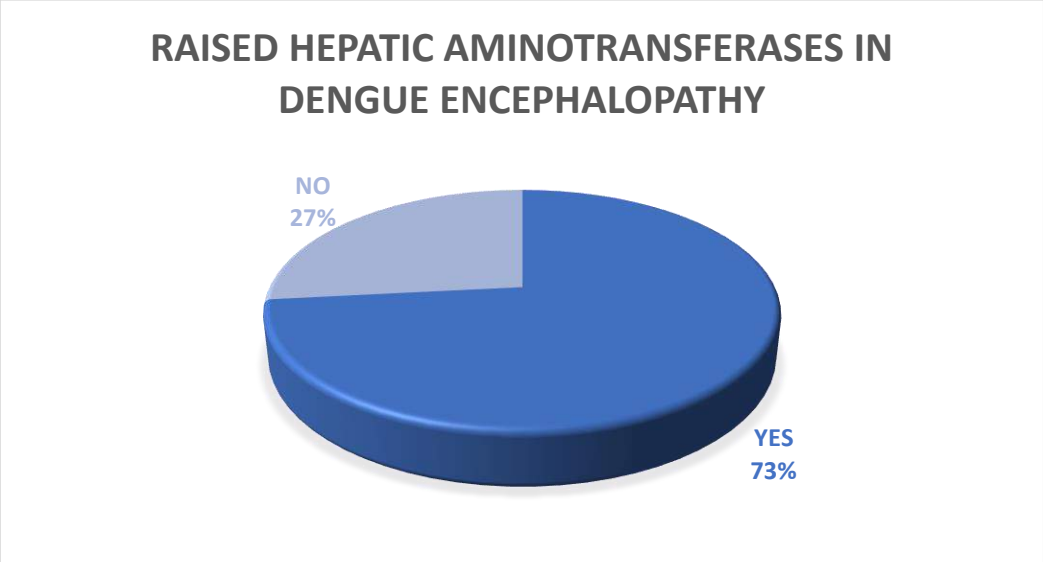


FIGURE 25: RAISED HEPATIC TRANSAMINASES IN PATIENTS WITH DENGUE ENCEPHLOPATHY

RADIOLOGICAL FINDINGS IN DENGUE RELATED NEUROLOGICAL COMPLICATIONS:

There were 3 patients in our study which involved brain parenchymal lesions, 2 of them had Haemorrhagic Encephalitis related to Dengue and 1 had features of Acute Disseminated Encephalomyelitis. Only 1 patient in this present study presented with ICH as neurological complication of Dengue.

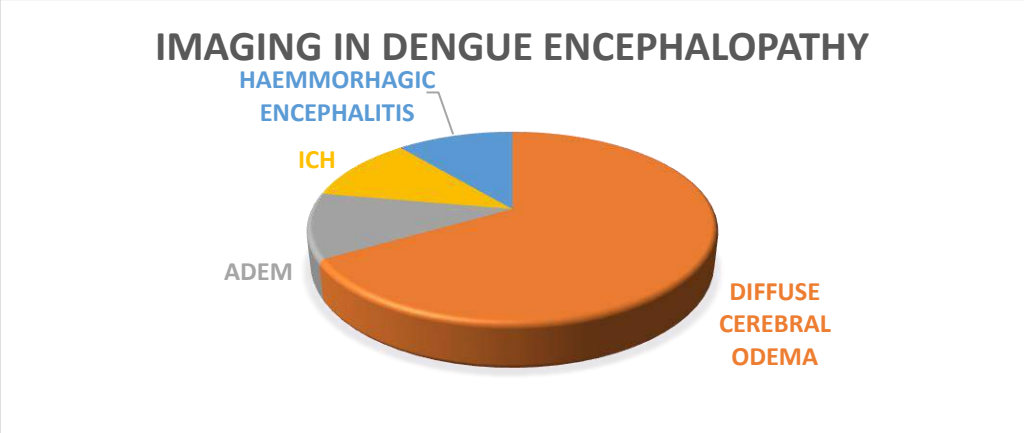


FIGURE 26: IMAGING IN DENGUE ENCEPHALOPATHY

DENGUE NEUROLOGICAL COMPLICATIONS CO RELATED WITH HYPOTENSION

Only 7 out of 30 patients with Dengue Encephalopathy/Encephalitis had Hypotension at the time of admission.

TABLE 44: DENGUE SHOCK SYNDROME AMONG PATIENTS WITH DENGUE ENCEPHALOPATHY

DENGUE ENCEPHALOPATHY	NO. OF PATIENTS (TOTAL -30)	%AGE
SHOCK PRESENT	6	20%
SHOCK ABSENT	24	80%

SCRUB RELATED ENCEPHALOPATHY:

In this study 5 out of 271 patients were diagnosed with Scrub associated Encephalopathy

AGE DISTRIBUTION

No age group predominance seen in this group of patients.

TABLE 45: AGE DISTRIBUTION OF PATIENTS WITH SCRUB ASSOCIATED ENCEPHALOPATHY

AGE GROUP	NO. OF PATIENTS (TOTAL-5)
18-40	2
>60	3

SEX DISTRIBUTION:

No gender predominance seen amongst this group of patients.

TABLE 46: SEX DISTRIBUTION OF PATIENTS WITH SCRUB ASSOCIATED ENCEPHALOPATHY

SEX DISTRIBUTION	NO. OF PATIENTS (TOTAL-5)
MALE	3
FEMALE	2

MRI FINDINGS:

MRI was s/o the following findings, all patients had Scrub IGM positivity

Table 47: MRI FINDINGS OF PATIENTS WITH SCRUB ASSOCIATED ENCEPHALOPATHY.

MRI FINDINGS	NO. OF PATIENTS(TOTAL-5)
MULTIFOCAL INFARCTS	1
HAEMORRHAGIC ENCEPHALITIS	1
MENINGO ENCEPHALITIS	1
NORMAL	2

CSF Findings:

Out of the 5 patients who were included in this group, CSF analysis was done in 3 patients and it was s/o lymphocytic predominant picture with low CSF glucose, High proteins and low/borderline CSF ADA levels.

LEPTOSPIRA RELATED NEUROLOGICAL COMPLICATIONS.

There were 3 patients out of 271 who were diagnosed with Leptospira related neurological complications. We were able to reach this diagnosis in view normal CT head and Normal MRI in all 3 patients with No abnormality in CSF in one patient and high TLC with lymphocytic predominance along with low CSF glucose and High CSF protein levels in second patient. In the third patient CSF could not be done.

POST COVID ENCEPHALOPATHY

In our study, post covid encephalopathy was a diagnosis of exclusion**.**

The major presenting symptoms were fever, state of confusion, drowsiness and one patient also had H/O seizures.

First patient (presented with abnormal body movements) had MRI s/o FLAIR hyperintensities and diffusion restriction throughout cerebral white matter, CSF analysis was unremarkable.

The second patient had MRI s/o transient splenial lesion likely secondary to hypoxia, CSF analysis was unremarkable.

In the third patient CT head was normal but since patient was intubated and was on inotropic support MRI Brain was not feasible, though CSF analysis was done which turned out to be normal.

CRYPTOCOCCAL MENINGITIS:

Amongst 271 patients, 4 patients were found to have acute (within 15 days) presentation of Cryptococcal Meningitis, all were HIV Negative.

All these patients were above the age of 50 years and 2 had TYPE 2 DM (uncontrolled status) with Diabetic kidney disease as accompanying co morbidity, one had H/O prolonged steroids in view of Obstructive airway disease and loss to follow up.

All these patients had history of fever with drowsiness,1 patient also had H/O abnormal body movements.

CSF analysis of all these patients was positive for India Ink and Cryptococcal Antigen was positive in 3. (One not sent)

MRI findings were s/o Leptomeningitis with Vasculitic infarcts in 2 patients. Both of them were given iv antifungals, they responded well and were discharged in stable condition. One patient had a normal MRI, treated with iv antifungals and had started responding but took DAMA due to monetary issues. One patient had e/o evolving brain abscess on MRI, got intubated, remained drowsy despite all treatment and eventually went into shock and expired.

OUTCOME:

In the given table the percentage of each outcome (recovery/ DAMA/ death) is calculated with total number of cases of the etiological entity as the denominator.

TABLE 48: OUTCOME OF EACH ETIOLOGICAL ENTITY AT THE TIME OF DISCHARGE (DIVIDED INTO RECOVERY/DAMA/DEATH)

	OUTCOME			
	RECOVERED (156)	**DAMA (48)**	**DEATH (67)**	**TOTAL (271)**
TBM	56(70.8%)	15(18.9%)	8(10.1%)	79
Viral meningo encephalitis	19(57%)	2(0.06%)	12(36.3%)	33
Dengue encephalopathy/ encephalitis	16(53%)	2(0.06%)	12(40%)	30
Pyogenic	6(50%)	3(25%)	3(25%)	12
Scrub related encephalopathy	4(90%)	0	1(0.20%)	5
Septic encephalopathy	46(46%)	24(24.2%)	29(29.3%)	99
Post covid encephalopathy	1(33.3%)	1(33.3%)	1(33.3%)	3
Leptospira induced encephalopathy	3(100%)	0	0	3
Cryptococcal meningitis	2(50%)	1(25%)	1(25%)	4

Here we observed that in patients with TBM, the proportion of patients who recovered out of total TBM patients was the highest than any other significant etiological entity. (70.8% of the total TBM patients recovered), though all 3 Leptospira associated encephalopathy patients improved, the data is too small to reach a definite conclusion of 100% recovery. It was found that patients with Dengue related neurological complications had the greatest number of deaths than any other etiological entity. (40% of the total Dengue Encephalopathy/Encephalitis died) followed by patients with SAE (29.3% of the total SAE patients died). All the three patients whose cause of Acute Febrile Encephalopathy remained undiagnosed, recovered.

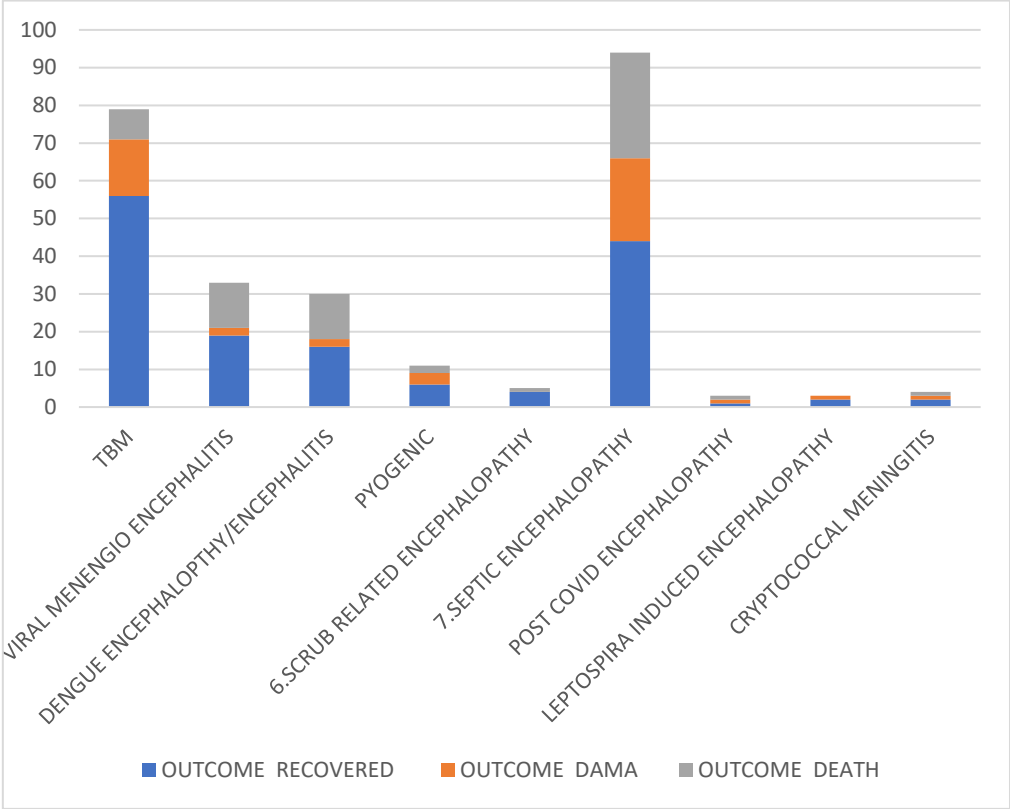


FIGURE 27: OUTCOME OF VARIOUS ETIOLOGICAL ENTITIES IN TERMS OF RECOVERY/DAMA/DEATH AT THE TIME OF DISCHARGE (total no. of cases of each etiological entity as denominator)

It was found in our study that out of the total recovered patients, the maximum recovery rate was found in patients with TBM (35.9%) followed by Sepsis Associated Encephalopathy (28.2%) despite the latter being the most common aetiology of AFE.

The total no. of patients who went for DAMA or expired were 115. Patients with Sepsis Associated Encephalopathy had the maximum no. of cases who either went DAMA/expired (46%). Majority of patients with SAE who went for DAMA were in shock/intubated and were expected to have a poor outcome.

IMPACT ON OUTCOME WHEN AGE GROUPS ARE COMPARED:

When different age groups were compared with each other in terms of outcome we did not receive results which are statistically significant, (p value :0.214) hence concluding that the age is not a statistically significant variable in the outcome of patients with acute febrile encephalopathy

TABLE 49: STATISTICAL INSIGNIFICANCE OF AGE AS A VARIABLE EFFECTING OUTCOME IN AFE

	OUTCOME						TOTAL	p-value
	RECOVERED (N=156)		DAMA (N=48)		DEATH (N=67)			
< 30	32	20.5%	5	10.4%	9	13.4%	46	0.214
31-50	35	22.4%	10	20.8%	20	29.9%	65	
51-70	55	35.3%	24	50.0%	20	29.9%	99	
> 70	34	21.8%	9	18.8%	18	26.9%	61	

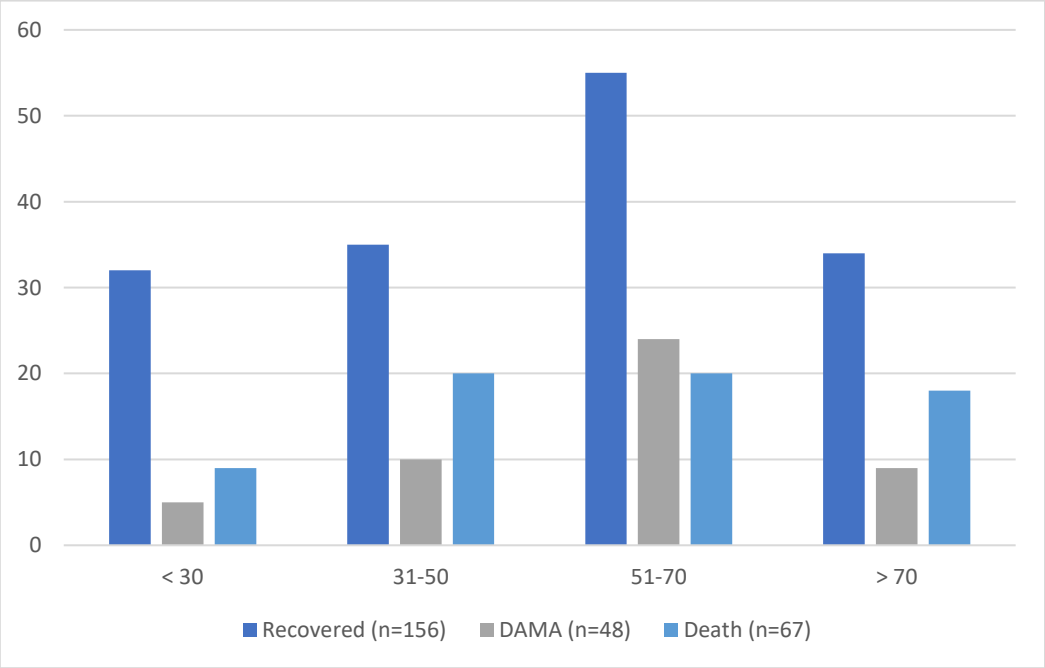


FIGURE 28: DIFFERENT AGE GROUPS COMPARED WITH EACH OTHER IN TERMS OF OUTCOME

IMPACT ON OUTCOME WHEN GENDER IS COMPARED

No statistical significance found in when gender was compared in terms of outcome.

TABLE 50: STATISTICAL INSIGNIFICANCE SHOWN WHEN GENDER WAS COMPARED IN TERMS OF OUTCOME

	OUTCOME						TOTAL	CHI-SQUARE VALUE	P-VALUE
	RECOVERED (N=156)		DAMA (N=48)		DEATH (N=67)				
F	61	39.1%	17	35.4%	27	40.3%	105	3.416	0.491
M	95	60.9%	31	64.6%	40	59.7%	166		

OUTCOME OF INDIVIDUAL ETIOLOGICAL ENTITIES:

1. MENINGITIS:

OUTCOME IN TUBERCULOUS MENINGITIS:

Out of total 79 patients who were diagnosed with TBM, 71% (56) recovered. (Maximum recovery rate amongst all other etiologies).

19% (15) took DAMA and 10% (8) of the patients died.

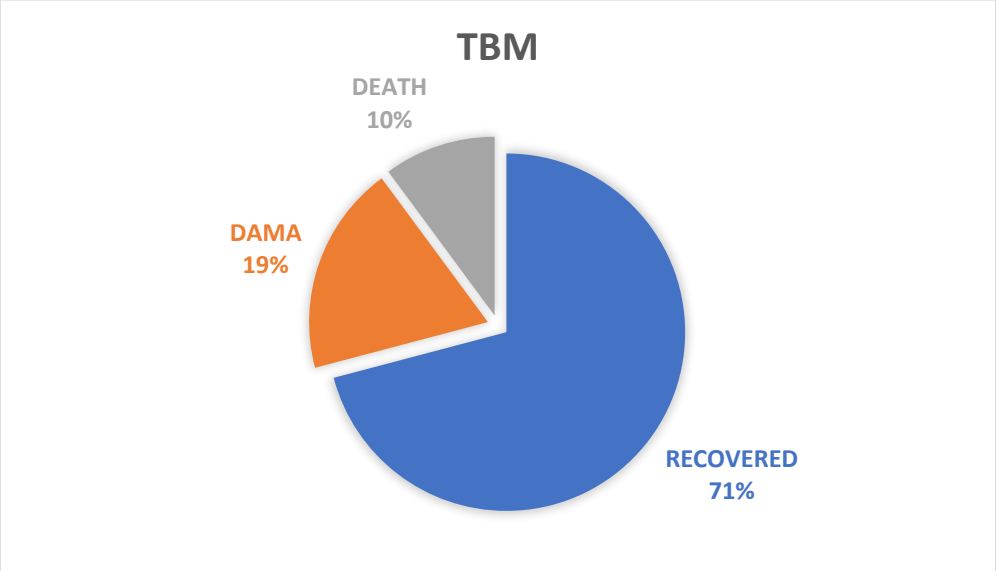


FIGURE 29: EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH TUBERCULOUS MENINGITIS.

STASTICAL SIGNIFICANCE OF MRI FINDINGS IN TBM

On evaluating for statistical significance of variables affecting outcome amongst patients with TBM, Hydrocephalus on MRI Brain and CT head was the most common finding amongst the patients who either dies or took DAMA mostly because of the requirement of prolonged stay and need for EVD in many cases. The p value in this case was 0.003.

TABLE 51: HYDROCEPHALUS ON RADIO IMAGING LEADING TO WORSE OUTCOME IN PATIENTS WITH TBM

	RECOVERED	DIED/ DAMA	TOTAL	P VALUE
MRI with hydrocephalus	10	14	24	
MRI without hydrocephalus	46	9	55	
Total	56	23	79	0.003

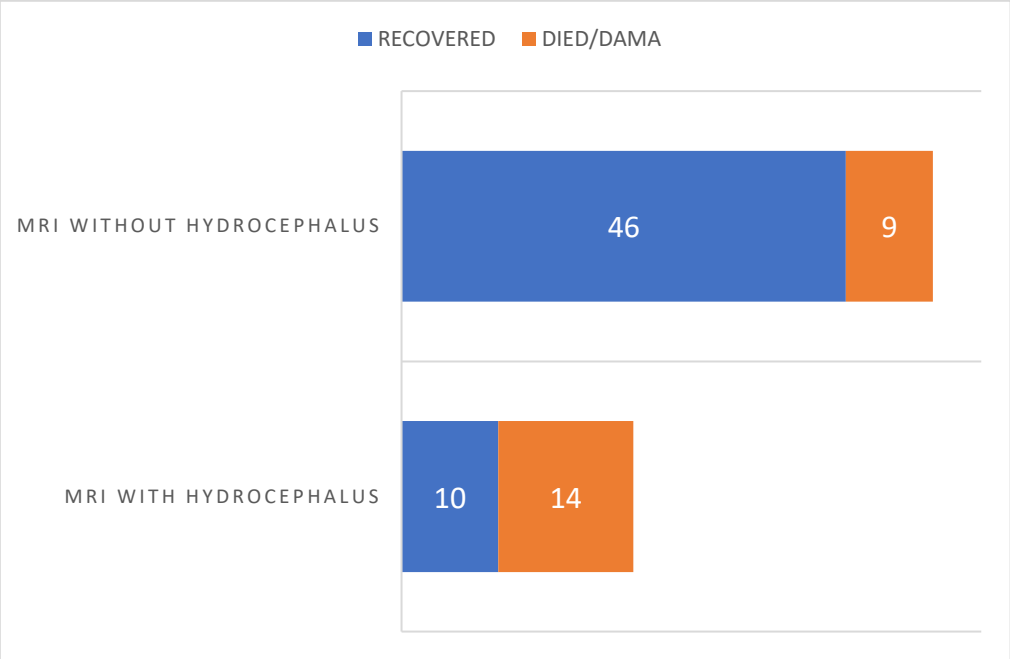


FIGURE 30: HYDROCEPHALUS ON MRI BRAIN LEADS TO ADVERSE OUTCOME

When other MRI findings like Leptomeningitis, Vasculitic infarcts and Encephalitis were compared with outcome, no statistical significance was found as mentioned below.

TABLE 52: LEPTOMENINGITIS ON MRI NOT AFFECTING OUTCOMEIN TBM

	RECOVERED	DIED/ DAMA	TOTAL	P VALUE
Leptomeningitis present	36	15	51	
No leptomeningitis	21	7	28	
Total	57	22	79	0.67

TABLE 53: VASCULITIC INFARCT ON MRI NOT AFFECTING OUTCOME IN TBM

	RECOVERED	DIED/ DAMA	TOTAL	P VALUE
Vasculitic infarct present	18	8	26	
No vasculitic infarct	39	14	53	
Total	57	22		0.68

STATISTICAL SIGNIFICANCE OF CSF VALUES IN TBM

When statistical significance was evaluated for CSF findings, the following results were obtained.

1. CSF GLUCOSE: When very low CSF glucose value(<35mg/dL) was compared with the outcome in terms of recovery/ death or DAMA, significant p value was obtained. Hence very low CSF glucose has an adverse effect on outcome in TBM.

TABLE 54: LOW GLUCOSE IN CSF EFFECTING OUTCOME IN TBM

	RECOVERED	DEATH/ DAMA	TOTAL	P VALUE
CSF Glucose Low	24	32	70	
CSF Glucose Normal	18	5	9	
Total	42	37	79	0.04

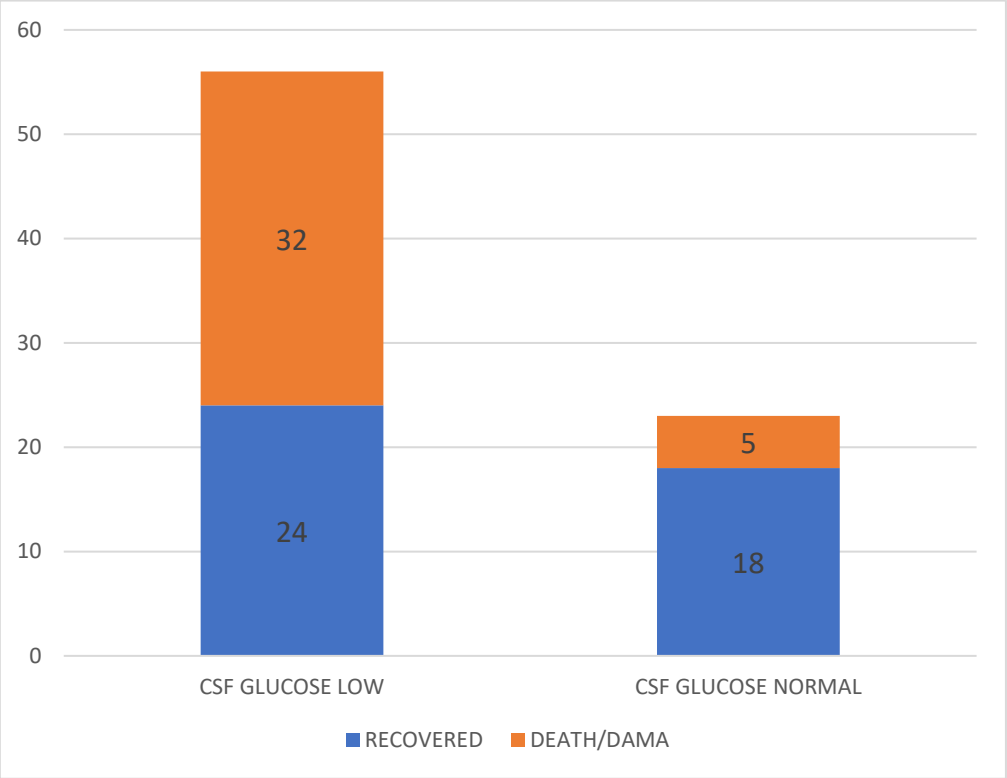


FIGURE 31: LOW GLUCOSE EFFECTING OUTCOME IN TBM

No other CSF value, high protein, high ADA or CBNAAT positivity had an impact on the outcome.

OUTCOME IN PYOGENIC MENINGITIS:

It was observed in our study that total number of patients with AFE who were diagnosed with Pyogenic Meningitis were 12 in number. Out of which 6 recovered (50%), 3 took DAMA (25%) and 3 died (25%).

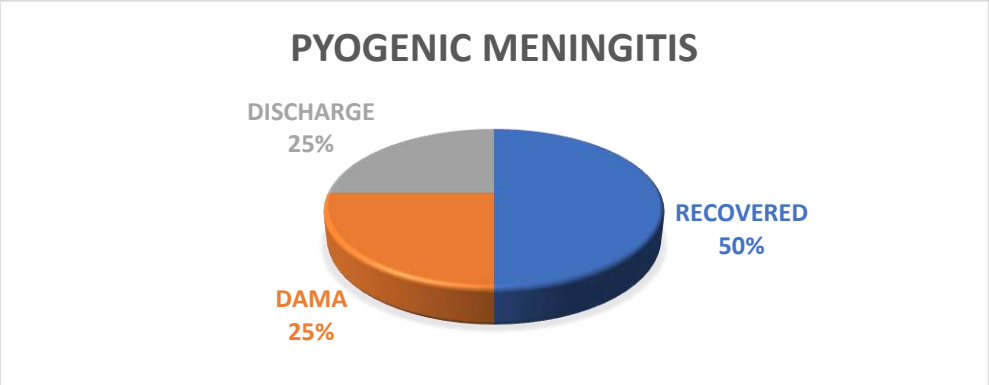


FIGURE 32: EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH PYOGENIC MENINGITIS

EFFECT OF LATEX ANTIGEN/CULTURE POSITIVITY IN CSF ON OUTCOME:

Out of 12 patients,3 had positive Streptococcus pneumoniae on Latex Antigen,1 patient had growth of Klebsiella in the CSF culture and one had growth of Salmonella typhi in CSF culture.

Out of these 5 patients, 3 expired and 2 recovered. There was no significant impact found of positive CSF growth on the outcome of the patients.

TABLE 55: CSF FLUID GROWTH HAS NO IMPACT ON OUTCOME

	RECOVERED	DAMA/ DEATH	TOTAL	P VALUE
CSF showing growth	2	3	5	
No growth on CSF	4	3	7	
TOTAL	6	6	12	0.55

4 out of 6 patients who either expired or went DAMA, had Leptomeningitis,2 had vasculitic infarcts and 1 had hydrocephalus on their MRI Brain, although MRI Brain findings had no statistically significant impact on outcome.

OUTCOME IN VIRAL MENINGITIS/MENINGO-ENCEPHALITIS:

In our study it was observed that out of the 33 patients who were diagnosed with Viral Meningo-encephalitis, 19(58%) recovered whereas 12 patients expired (36%) and 2 patients went DAMA (6%).

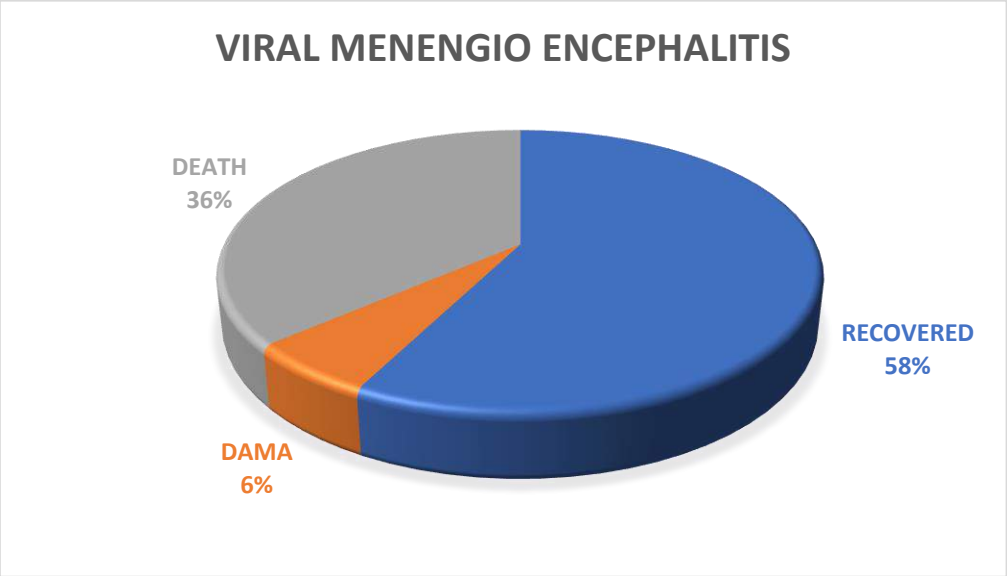


FIGURE 33: EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH VIRAL MENINGITIS/ MENINGO-ENCEPHALITIS.

SIGNIFICANCE OF HSV PCR POSITIVITY IN OUTCOME:

When HSV PCR positivity was compared to the outcome, no statistical significance was found.

TABLE 56: STATISTICAL INSIGNIFICANCE WHEN HSV RNA PCR POSITIVITY IN CSF WAS COMPARED WITH THE OUTCOME.

HSV PCR	RECOVERED		DAMA/ DEATH		TOTAL	P VALUE
	136	87.2%	102	212.5%	238	0.142
NEGATIVE	10	6.4%	7	14.6%	17	
POSITIVE	10	6.4%	6	12.5%	16	

SIGNIFICANCE OF MRI FINDINGS IN OUTCOME:

When those patients who had evidence of Brain parenchymal involvement were compared with those who did not, there was no statistical difference between these two groups in terms of outcome.

TABLE 57: NO AFFECT OF ENCEPHALITIS ON MRI ON OUTCOME OF PATIENTS WITH VIRAL ENCEPHALITIS

MRI Findings	RECOVERED	DEATH/ DAMA	TOTAL	P VALUE
Encephalitis	11	8	19	
No Encephalitis	8	6	14	
Total	19	14	33	0.96

EFFECT OF HYPOTENSION AND POOR GCS ON OUTCOME OF VIRAL MENINGO-ENCEPHALITIS:

In our study it was found that the patients presenting with hypotension and those who require intubation have a poor outcome as compared to normotensive patients with better GCS.

TABLE 58: HYPOTENSION LEADING TO ADVERSE OUTCOME IN PATIENTS WITH VIRAL MENINGIO-ENCEPHALITIS.

	RECOVERED	DEATH/ DAMA	TOTAL	P VALUE
Hypotension	3	9	12	
Normotension	16	5	21	
Total	19	14	33	0.04

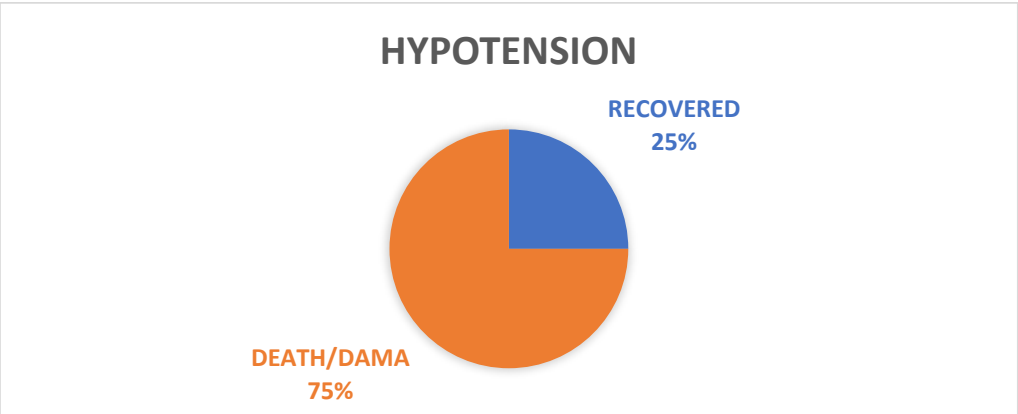


FIGURE 34: HYPOTENSION LEADING TO ADVERSE OUTCOME IN PATIENTS WITH VIRAL MENINGIO-ENCEPHALITIS

OUTCOME IN DENGUE ENCEPHALOPATHY/ENCEPHALITIS:

In our study it was found that there were 30 patients who were diagnosed with neurological complications of Dengue. Out of which 16 patients (53%) recovered, 12 (40%) expired and 2(7%) patients took DAMA. The 2 patients who took DAMA were both on mechanical ventilatory support.

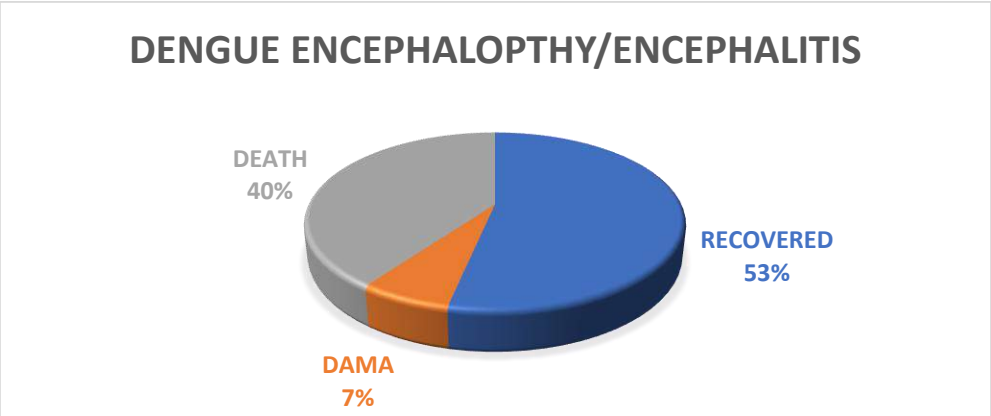


FIGURE 35: EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH DENGUE ENCEPHALITIS/ENCEPHALOPATHY

EFFECT OF RAISED AMINOTRANSFERASES ON PATIENTS WITH DENGUE RELATED NEUROLOGICAL COMPLICATIONS:

22 out of total 30 patients had significantly raised aminotransferase levels (above 5 times the upper normal limit),13 out of these 22 patients (59%) with raised SGOT and SGPT either died/went for DAMA. But when compared with the group not showing transaminitis, no statically significant effect on the outcome was observed.

TABLE 59: NO EFFECT OF TRANSAMINITIS ON OUTCOME OBSERVED.

	RECOVERED	DAMA/ DEATH	TOTAL	P VALUE
HIGH SGOT/SGPT	10	12	22	
NORMAL SGOT/PT	6	2	8	
TOTAL	16	14	30	0.15

EFFECT OF HYPOTENSION ON OUTCOME OF DENGUE ENCEPHALOPATHY PATIENTS

It was found that in our study,6 out of 7 patients with Shock on presentation expired (86%). When hypotensive patients were compared with normotensive patients in terms of outcome, significant p value was obtained, hence Hypotension is a significant variable and is related to adverse outcome.

TABLE 60: HYPOTENSION EFFECTING OUTCOME IN DENGUE ENCEPHALOPATHY PATIENTS

	RECOVERED	DAMA/ DEATH	TOTAL	P VALUE
HYPOTENSION	1	6	7	
NORMOTENSION	15	8	23	
TOTAL	16	14	30	0.01

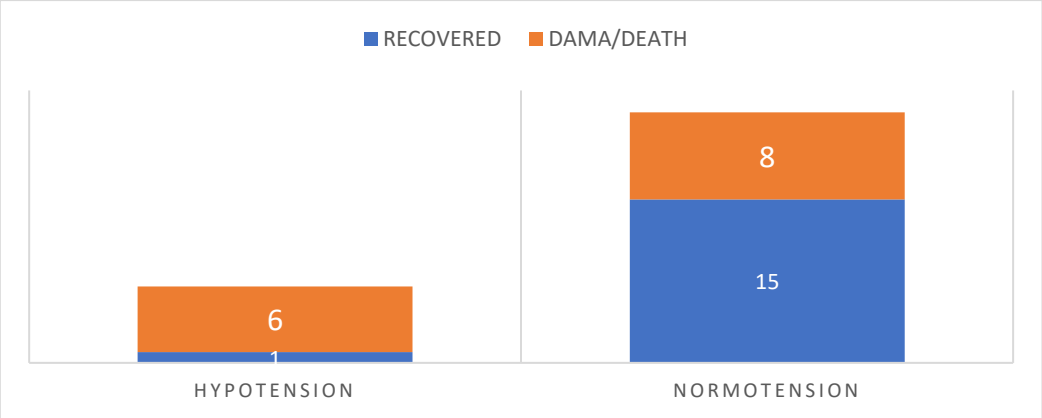


FIGURE 36: HYPOTENSION LEADING TO ADVERSE OUTCOME IN DENGUE ENCEPHALOPATHY

No significant effect on outcome was obtained when the patients having positive MRI /CT findings were compared with normal Neuroimaging. In the present study there were 2 patients who presented with Hemorrhagic Encephalitis as a complication of Dengue, both of which survived but had a prolonged hospital stay and both were discharged on tracheostomy support with GCS of E4VTM6.

One patient who developed ICH as a complication of Dengue did not survive.

Also, evidence of Polyserositis, hemoconcentration was associated with Dengue shock and hence poor outcome of Dengue related neurological outcomes.

OUTCOME IN SEPSIS ASSOCIATED ENCEPHALOPATHY:

SAE had been found as the most common etiology in patients with Acute Febrile Encephalopathy (99 out of 271 patients). Out of them 46% patients recovered,24% of the patients took DAMA and 30% died.

This group had the maximum mortality rate amongst all other groups (29 out of 67: 43.2%)

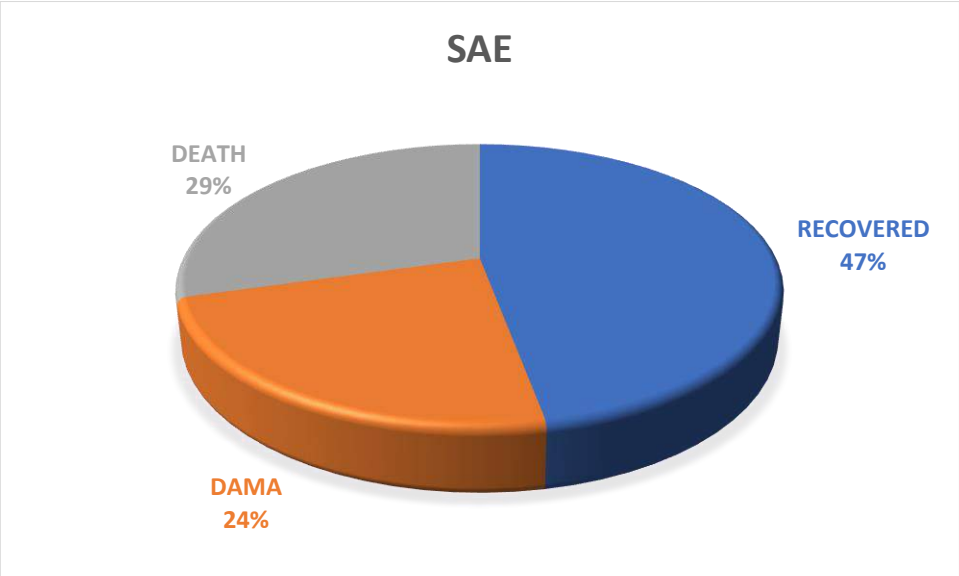


FIGURE 37: EACH OUTCOME SEPARATELY COMPARED IN PATIENTS WITH SEPSISASSOCIATED ENCEPHALOPATHY

IMPACT OF TYPE 2 DM IN OUTCOME OF PATIENTS WITH SAE

Out of 50 patients with TYPE 2 DM and SAE, 31 patients were sent DAMA/Expired (62%), 19 patients recovered.

TABLE 61: OUTCOME OF PATIENTS OF SAE WITH TYPE 2 DM

SEPSIS ASSOCIATED ENCEPHALOPATHY WITH TYPE 2 DM		
RECOVERED	17	34%
DAMA/EXPIRED	33	66%
TOTAL	50	100%

But when statistical significance of the effect of TYPE 2 DM on the outcome of patients with Acute Febrile Encephalopathy was evaluated, the p value came out to be 0.055, hence not significant.

TABLE 62: IMPACT OF TYPE 2 DM ON OUTCOME OF SAE

	RECOVERED	DAMA/ DEATH	TOTAL	P VALUE
DIABETIC	17	33	50	
NON DIABETIC	26	23	49	
TOTAL	43	56	99	0.055

IMPACT OF SEPTIC SHOCK ON OUTCOME OF SAE:

Out of 57 patients who presented in septic shock 42(73.9%) patients either expired/ went DAMA, mostly within 24 hours of admission.

And when compared to patients with organism growth in their body fluids, out of 24 such patients who presented in septic shock, 20 (83.3%) people either expired or took DAMA.

TABLE 63: SIGNIFICANT IMPACT OF SEPTIC SHOCK ON OUTCOME OF SAE

	RECOVERED	DAMA/ DEATH	TOTAL	P VALUE
HYPOTENSION	15	42	57	
NORMOTENSION	31	11	42	
TOTAL	46	53	99	0.001

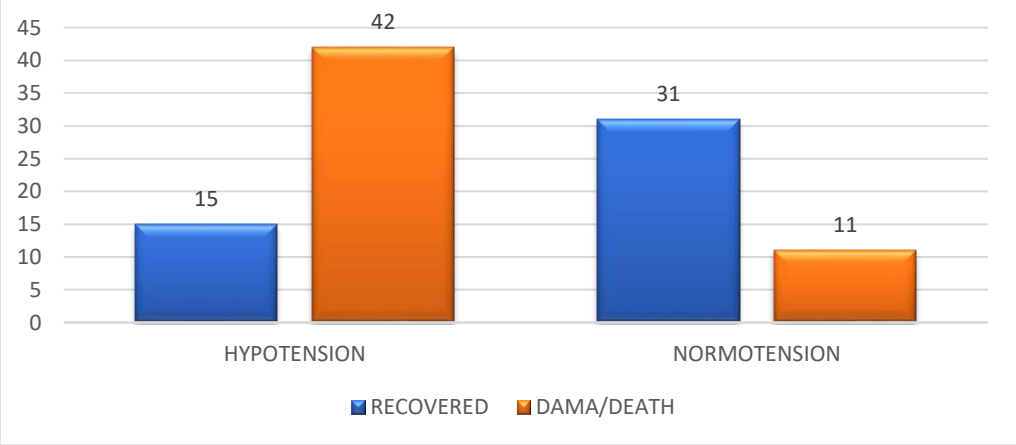


FIGURE 38: SIGNIFICANT IMPACT OF SEPTIC SHOCK ON OUTCOME OF SAE

EFFECT OF ANEMIA ON OUTCOME:

Hb<8 g/dL was taken as the cut off for anaemia. 34 out of 99 patients had anaemia and out of then 20 patients either expired or took DAMA, but no statistically significant impact found on outcome.

TABLE 64: EFFECT OF ANEMIA ON OUTCOME IN SAE

PALOR	RECOVERED	DAMA/DEATH	TOTAL	P VALUE
PRESENT	14	20	34	
ABSENT	32	33	65	
TOTAL	46	53	99	0.58

EFFECT OF SEPSIS RELATED DERANGED RENAL AND HEPATIC FUNCTION ON OUTCOME:

71 out of 99 patients had evidence of derangement of either renal or hepatic or both organ systems evident in form of raised serum creatinine and serum bilirubin. 38 out of these had an adverse outcome.

TABLE 65: EFFECT OF RENAL/HEPATIC DERANGEMENT ON OUTCOME IN PATIENTS WITH SAE

DRANGED RFT/LFT	RECOVERED	DAMA/ DEATH	TOTAL	P VALUE
YES	33	38	71	0.01
NO	21	7	28	
	54	45	99	

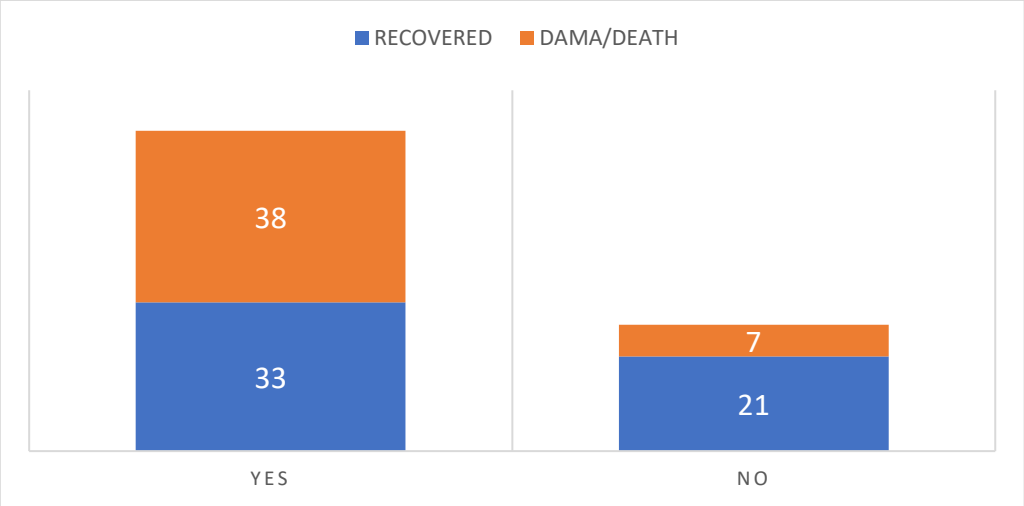


FIGURE 39: EFFECT OF RENAL/HEPATIC DERANGEMENT ON OUTCOME IN PATIENTS WITH SAE

OUTCOME IN CRYPTOCOCCAL MENINGITIS:

MRI findings were s/o Leptomeningitis with Vasculitic infarcts in 2 patients. Both of them were given iv antifungals, they responded well and were discharged in stable condition. One patient had a normal MRI, treated with iv antifungals and had started responding but took DAMA due to monetary issues. One patient had e/o evolving brain abscess on MRI, got intubated, remained drowsy despite all treatment and eventually went into shock and expired.

OUTCOME OF SCRUB TYPHUS/LEPTOSPIRA ASSOCIATED /POST COVID NEUROLOGICAL COMPLICATIONS:

In this study, it was found that 5 out of 271 patients had Scrub typhus associated Encephalopathy. Out of these 5,4 patients recovered and 1 patient expired (due to overlying sepsis and septic shock post developing VAP).

Leptospira associated neurological complications were found in 3 patients, all 3 of which recovered without residual neurological complications.

Amongst Post covid Encephalopathy patients (3 out of 271), one patient recovered with residual delirium, one patient went DAMA due to prolonged dependence on mechanical ventilatory support and one patient expired due to overlying complications.

There was no significant co relation of positive or particular Radio-imaging findings or CSF analysis report with the outcome of these patients.

The data in this study cannot be used to reach appropriate deductions since the sample size of this group of patients is very small.

DISCUSSION

This study was conducted in a tertiary care center in North India and included all the patients with Acute Febrile Encephalopathy who were 18 years and above and had no other (metabolic/ cerebrovascular accident that independently explained the presentation) etiology for their presentation. A total of 271 patients were enrolled in the study and all of them fulfilled the inclusion criteria. The observations, results and statistical data were compared with previously published studies done on similar lines. The data compiled and the results obtained have been discussed below.

ACUTE FEBRILE ENCEPHALOPATHY

It was observed that in our study, out of 271 patients, maximum number of patients belonged to the age group of > 60years (40.5%). The mean age in our study was 51.95 ±19.2 years. In the study by Modi et al [1] the age of patients ranged from 13 to 70 years with a mean of 31.89 ± 14.24 years. In the study by Bhalla et al [110] a total of 123 patients were studied, all were above 12 years of age the mean age was 30.14±14.79 years. In the study by Job et al [120] in which 265 patients, their age ranging from 15 to 86 years. The mean (SD) age was 46.9 (19.8) years.

In our study, out of 271patients, 166 (61.3%) were male and 105 (38.7%%) were female. Male preponderance was observed with male: female ratio was 1.58:1. In 2012, Modi et all [1] did a study in patients with similar presentation and found that there were 76 (63.3%) males and 44 (36.7%) females. In 2014 Bhalla et all [110] did a study on Acute Febrile Encephalopathy where there was a male preponderance with 78.1% patients being male in their study group. In 2015 Reza et all [111] did a study, 100 patients aged ≥65 years with fever and impaired consciousness were included. Of the studied population, 61(61%) were male and 39 (39%) were female, again highlighting the male preponderance. In 2016 Job et al [120] did a study including 265 patients and found that the sex ratio in the cohort was almost equal (111, 58% male). Hence in 4 out of 5 studies (including the present study) it was

seen that the males are more prone to develop Acute Febrile Encephalopathy than the females. Although none of the CNS infections are known to have a male predominance, this apparent male predominance can be attributed to the male dominated social system where a sick male gets preferential medical attention.

In our study, 89 patients (32.8%) had underlying Diabetes Mellitus. Hypertension was present in 53 patients (19.6%). 11 patients had CAD, 11 were hypothyroid, 3 had HIV co infection, 12 were bedridden,8 had cancer and 12 had old healed pulmonary Koch's/ Miliary Tuberculosis. In the study by Reza et al [111], the frequency of underlying diseases showed that of all included participants, 46 patients had hypertension, 40 patients had a chronic neurological disorder, 27 had diabetes mellitus and 25 patients suffered from ischemic heart disease. In addition, 19 patients were in bed ridden status, 5 patients had secondary immune deficiency and there were 4 cases of cancer. Job et al [120] study group contained Diabetes in 80 (30.2%) and hypertension in 61 patients (23%) as the major comorbidities. In the present study, statistical analysis showed that no significant difference exists between any of the underlying medical conditions and CNS-associated and CNS unassociated encephalopathy, as well as infectious and non-infectious encephalopathy.

In the present study all 271 patients fulfilled the criteria of documented fever within 15 days of presentation accompanied with or followed by altered sensorium. Other than that, Headache (18.5%) was the most common presenting complaint along with vomiting (18.1%) and abnormal body movements (17.7%). Neck rigidity was present in15.3% of patients. In the analysis by Job et al[120] major symptoms noted were altered sensorium, headache, nuchal rigidity, vomiting and seizures. Modi et al (2012) [1] concluded that the most common complaints were fever (100%), headache (100%), and altered mental state (100%). In the study by Bhalla et al in 2014[110] fever and headache were the most common symptoms seen in 100% and 94% patients, respectively. 26%

of the patients had seizures at presentation and Neck stiffness was present in 26% patients. The mean GCS score of the 271 patients enrolled in our study was 10.44 (SD 2.44). In the study by Modi et al [1] the Glasgow Coma Scale (GCS) score at the time of presentation was ≤7 in 21 (17.5%) patients, while 99 (82.5%) patients had a GCS of >7 and in the study by Job et al [120] mean Glasgow Coma Scores (GCS) at admission was 11.8 (SD 3.2).

In the present study it was observed that Palor was the most common sign noticed amongst the patients enrolled in the study when GPE was done. Also, palor has a significant impact on outcome of the patients. 57 out of 271 patients enrolled in the study had palor, 23 out of them recovered while rest took DAMA/Discharge.

On systemic examination, it was observed that although positive findings in abdomen, respiratory system, cardiovascular system had a co relation with outcome in Sepsis Associated Encephalopathy but overall systemic examination was not s/o of a significant co relation with outcome in patients with AFE.

In our study it was found that SAE was the most common etiology of acute febrile encephalopathy (36.5%) followed by TBM (29.2%) and Viral meningitis/ meningo-encephalitis (12.2%).Dengue associated Encephalopathy/ Encephalitis was found in 11.1% patients. Pyogenic Meningitis was found amongst 4.4% cases. 5 patients had Scrub related Encephalopathy, 4 presented with Cryptococcal Meningitis and 3 had Leptospira related aseptic meningitis, 3 were found to be Post Covid Encephalopathy after ruling out all other causes.3 patients remained undiagnosed despite extensive imaging, all serologies and CSF analysis. In the study by Bhalla et al [110] a total of 42 patients had meningitis with 32 patients (25.2%) having acute pyogenic meningitis and 10 patients (7.87%) having tuberculous meningitis (TBM). Thirty-eight patients (29.9%) had evidence of meningoencephalitis, which was followed by cerebral malaria, leptospirosis, and brain abscess as a cause of primary CNS infection. Sixteen patients (12.7%) had infections

elsewhere and were diagnosed as sepsis-associated encephalopathy (SAE). Four patients (3.15%) were diagnosed as having cerebral malaria and 3 patients each (2.36%) had leptospirosis and acute disseminated encephalomyelitis. However, Modi et al [1] found that in their study, Pyogenic Meningitis was the most common cause. (36.7%), followed by viral meningo-encephalitis (28.35), cerebral malaria (21.7%), SAE was only found in 9.17% AND TBM in 4.2%. Reza et al [111] concluded that out of the 100 above 65 years of age patients, participating in their study, the aetiology of febrile encephalopathy in 94 cases including 10 cases with intra-cranial pathology (10%) was infectious. Direct CNS involvement was ruled out in all other 84 cases (including one with disseminated tuberculosis and one with systemic brucellosis). In 6 cases, non-infectious conditions (6%) caused fever and loss of consciousness. In the study by Job et al,[120] Viral etiology was found to be the most common followed by TBM, Pyogenic meningitis and other non-infectious etiology. Tuberculous meningitis (TBM) although infrequently reported in other series was a predominant cause of AFE in their study and even our study reflecting the high prevalence of TB in India and better access to diagnostics such as TB polymerase chain reaction (GeneXpert MTB/RIF) and liquid culture method (MGIT) that makes early diagnosis feasible. Evidence of tuberculosis from lymph node or bone marrow aids in the diagnosis.

In our study SAE was diagnosed more and had higher mortality as well probably because it is a referral center for nearby districts, states and patients are already in a refractory shock state on admission.

It is postulated that alteration in sensorium in a patient with CNS infection indicates an element of parenchymal involvement. [109,121] This can explain the altered mentation in meningoencephalitis patients. The reason for altered sensorium in meningitis is postulated to be the spillage of inflammatory cells to the adjacent brain parenchyma and the resultant parenchymal involvement.[121] In SAE, metabolic alterations and

inflammatory cytokines may play an important role in the pathogenesis of encephalopathy rather than direct parenchymal involvement.

Though dengue was earlier thought to be a non-neurotropic virus, nowadays dengue encephalopathy is a well-recognized and common entity, The possible mechanisms are liver failure (hepatic encephalopathy), cerebral hypoperfusion (shock), cerebral edema (vascular leak), deranged electrolytes, and intracranial bleeding due to thrombocytopenia or coagulopathy, which is secondary to hepatic failure.[122] There are subsets of patients in whom the cause for neurological injury remains unclear, even after excluding the above-mentioned indirect mechanisms. Dengue neurotropism has been suggested as the possible mechanism in such cases because of evidence of dengue virus and dengue IgM antibodies has been discovered in CSF of encephalopathy patients, which suggests that the dengue virus is capable of CNS infection [75].

In the present study 153 out of 271 patients underwent CT Head on admission and 228 patients got an MRI Brain done at least once during hospital stay. Hydrocephalus was the most common pathological finding in CT head (23 out of 153 scans) and mostly associated with TBM (23 out of 25). Diffuse Cerebral oedema reflecting a metabolic insult was the most common finding of Dengue Encephalopathy (5 out of 6) and one out of the 2 patients with Dengue Hemorrhagic Encephalitis got a CT head done which was s/o multiple small hemorrhages.

Amongst findings of MRI Brain, Leptomeningitis was the most common etiology which was found in TBM patients in maximum numbers (51 out of 59).33 patients had vasculitic infarcts (Most common: TBM) and scan of 32 patients showed Encephalitis evidence (Most common: Viral). 1 patient with SAE had findings s/o PRES, 1 patient of Dengue Encephalitis had features of ADEM and 1 patient with Dengue Hemorrhagic manifestations had an ICH evident on MRI Brain.
In TBM, CBNAAT positive CSF was found in 33% of the patients (26 out of 79) and CSF ADA (sensitivity in this study:90.4%) and Low CSF

glucose (sensitivity in this study:92.3%) had been established as significant surrogate markers for diagnosis of TBM in absence of CBNAAT positivity along with high CSF protein. Most of the patients (53 out of 79) were diagnosed on the basis of low CSF sugar, High CSF protein and High CSF ADA levels. Leptomeningitis was the most common MRI Finding and Tuberculomas were the most specific finding. Etiology of altered sensorium is postulated to be Meningeal inflammation, vasculitic infarcts, brain parenchymal involvement, hydrocephalus in cases of TBM.

In Pyogenic Meningitis, 3 patients had Latex Antigen positivity, 1 had growth of Klebsiella in CSF culture and 1 had growth of S.typhi in their CSF fluid. Other 7 patients were diagnosed on the basis of neutrophilic predominance and low CSF glucose and high CSF protein levels. MRI findings included Leptomeningitis, vasculitic infarcts, meningo encephalitis and hydrocephalus.

In Viral Meningitis/Meningoencephalitis, abnormal body movements were most frequently found as a presenting complaint. Though CSF Viral panel was an option available at nearby centers but the cost is a limiting factor, hence in suspected patients, mostly HSV PCR was sent and 16 out of 33 were positive. In the remaining patients diagnosis of Viral Meningitis/Meningoencephalitis was formulated on the basis of either MRI findings or CSF findings s/o high protein, normal glucose levels and clinical presentation. Patients with Herpes Encephalitis may have specific MRI findings s/o bilateral asymmetrical involvement of limbic system, including medial temporal lobes, insular cortices and inferolateral frontal lobes and cingulate gyri. (found in 17 out of 33 patients in our study).

In SAE, elderly age group(>65years) and male predominance were a significant finding. 59 out of 99 patients had organism growth in samples of their cultures and urine was found to be the most common media for this growth and E.coli, the most common organism. High serum Procalcitonin is an e/o Bacterial Sepsis and was found significant in 64

out of the total 99 patients who were diagnosed with SAE. Patients with Sepsis Associated Encephalopathy were diagnosed after excluding other possible etiologies. Patients who came to Emergency Department in septic shock did get a CT head done at least for ruling out intracranial bleed or structural changes but further investigations were not possible in many patients in view of unstable clinical status or early mortality. In these patients diagnosis of SAE was made if the initial evaluation was s/o any evidence of sepsis (growth in culture/pus cells in urine routine, high TLC, high Procalcitonin, Leucopenia, history of high grade fever, imaging findings suggesting source of sepsis etc.) along with signs of encephalopathy. In many patients CSF analysis and further neuroimaging in form of MRI Brain was possible. In all the patients with SAE who got their CSF analysis done, it came out to be normal or there was mild raise in CSF protein levels. Brain imaging and CSF analysis were mostly within normal limits in patients with SAE which aided in the diagnosis. Some important MRI findings did include septic emboli, cytotoxic oedema, leukoencephalopathy and PRES, though very rare.

In Dengue related neurological complications, raised aminotransferase levels were a significant co relation (22 out of 30) but had no statistically significant impact on outcome. Most common neuro imaging finding was Diffuse cerebral oedema but 2 patients did have e/o Hemorrhagic Encephalitis in their MRI Brain.

Scrub typhus as the etiology of neurological complications was a diagnosis of exclusion, CSF was s/o lymphocytic predominance with borderline ADA in most patients, CSF for Scrub IgM was not done in any patient and Scrub typhus IgM positivity in serum with neurological features was considered as sufficient evidence. MRI findings were S/o Hemorrhagic Encephalitis in one patient, vasculitic infarct in one patient and meningo-encephalitis in one.

The overall outcome at discharge of various etiological entities has been discussed in Table 48. It was found that the proportion of patients who recovered out of total TBM patients was the highest than any other

common etiological entity. (70.8% of the total TBM patients recovered). Patients with Dengue related neurological complications had the greatest number of deaths than any other etiological entity. (40% of the total Dengue Encephalopathy / Encephalitis died) followed by patients with SAE. Out of 67 patients who died, 29 were diagnosed with SAE and related septic shock, (43.2%-maximum mortality rate). Age, gender did not have any significant impact on outcome. Patients with TBM who had radiological e/o Hydrocephalus tend to have a worse outcome. Low CSF glucose (<35 mg/dL) was also related with adverse outcome. In Pyogenic Meningitis, Out of 12 patients, 6 patients recovered (50%), 3 took DAMA (25%) and 3 died (25%). In Viral Encephalitis, patients HSV RNA PCR in CSF did not have an impact on outcome of disease. Neither specific MRI findings of Herpes Encephalitis had an impact on the outcome of disease. Though patients who presented with hypotension or required mechanical ventilation did have a poor outcome.

Patients diagnosed with Dengue related complications had a direct co relation of Dengue shock syndrome with poor outcome. Though other findings like raised aminotransferases signifying liver injury did not have a significant impact on outcome.

In patients with SAE, hypotension was the major factor effecting outcome of the patient. Though Type 2 DM was most commonly present in patients with SAE but there was no significant co relation of presence of Diabetes and mortality. Sepsis related Multiple Organ Dysfunction Syndrome evident in form of deranged LFT'S, deranged RFT'S, low platelet count was associated with a significantly adverse outcome in patients with Sepsis Associated Encephalopathy.
In all the remaining etiological entities the sample size was too small to make adequate conclusions from the results obtained.

Acute Febrile Encephalopathy is a clinical spectrum involving patients presenting with fever and altered sensorium. In the present study, as compared to earlier studies where Pyogenic Meningitis, Viral Meningo Encephalitis or Tuberculous Meningitis had been established as the

most common etiologies, it was found that Sepsis Associated Encephalopathy is the commonest cause of such presentation followed by Tuberculous Meningitis. Hence highlighting the importance of keeping these differentials in mind during initial evaluation of these patients. This changing trend also points towards increasing complications due to sepsis in view of emergence of drug resistant strains of the pathological organism. Higher incidence of TBM in the study signifies high prevalence of TB in India and better access to diagnostics such as TB polymerase chain reaction (GeneXpert MTB/RIF) and liquid culture method (MGIT) that makes early diagnosis feasible.

LIMITATIONS:

1. Single centred study.
2. Patients less than 18 years not included.
3. Data on rare etiologies was not enough to draw satisfactory conclusions.
4. No long term follow up of the patients was done.
5. Definitive outcome of the patients who went for DAMA could not be documented.

SUMMARY AND CONCLUSIONS

1. We studied the etiological profile and outcome of 271 patients who presented to the Hospital with fever of less than or equal to 15 days duration which was accompanied or followed by altered sensorium.
2. Most of the patients in our study group were in the age group above 60 years (40.5%) with mean age 53.31 years ± 19.41 years.
3. Male predominance (61.3%) was seen with male:female ratio 1.58:1.
4. The common presenting complaint apart from fever and altered sensorium were headache (18.5%), vomiting (18.1%) and abnormal body movements. (17.5%)
5. The most common co- morbidity found in this study group was Type 2 DM (32.8%), and Palor was the most common sign observed on General Physical examination.
6. The most common etiology observed out of 271 patients in our study group was Sepsis Associated Encephalopathy (36.5%) followed by Tuberculous Meningitis (29.2%) and Viral Meningitis/Meningo-encephalitis (12.2%).
7. Hydrocephalus was the most common CT finding among patients with infectious CNS etiologies and Leptomeningitis was the most common MRI finding among them.
8. Leptomeningitis, Vasculitis, Ring enhancing lesions, Hydrocephalus were most commonly found in TBM whereas features of Encephalitis were most common found in Viral etiologies.
9. Amongst patients with TBM, 33% had CBNAAT positivity in CSF and significant CSF ADA, Low CSF glucose and High CSF Proteins were established as markers with high sensitivity in establishing the diagnosis of TBM.
10. Pyogenic Meningitis was only found in 4.4% (12 out of 271) of the patients included in this study, 5 were diagnosed on basis of Latex Antigen / CSF C/S positivity and 7 on basis of CSF findings.
11. Viral Meningitis/ Meningo-encephalitis was found in 12.2 % of patients based on HSV PCR positivity, MRI findings, CSF findings or clinical picture.

12. Among patients with SAE, elderly age group (>60years) and male predominance was present and Type 2 DM was the most common co-morbidity. In almost 85% of these patients the source of sepsis was identifiable on the basis of C/S growth in various sites, high PCT, presenting features or imaging findings pointing towards the source.
13. 60% of these patients were observed to have an organism growth in the cultures of their samples, out of which Urosepsis was the most common infection site and E. coli was the most common organism growth obtained.
14. Dengue related neurological complications were found in 11.1% of the patients. Higher hepatic transaminases had a significant co relation with Dengue complicating into neurological manifestations.
15. Scrub related neurological complications were found in 5, Leptospira related meningitis was found in 3, Post covid Encephalopathy was found in 3 and Cryptococcal Meningitis with acute presentation was found in 4 patients.
16. TBM had maximum recovery rate (70.8%) and most patients with Sepsis Associated Encephalopathy (53%) either expired or went DAMA mostly due to expected poor outcome.
17. It was found that neither age nor sex had an impact on outcome.
18. In TBM, Hydrocephalus on radio imaging and low CSF glucose was found to be related with adverse outcome.
19. Hypotension during any time during the course of hospitalisation was related with adverse outcome in Dengue/ Sepsis or Viral etiologies.
20. Deranged Hepatic and Renal function in patients with SAE along with Septic shock was related to adverse outcome.

CONCLUSION

Acute Febrile Encephalopathy is a broad term inclusive of many variable etiologies from direct CNS infection like TBM, Pyogenic Meningitis, Viral Encephalitis/Meningitis, Cryptococcal Meningitis to systemic diseases altering the CNS function by alternative mechanisms like direct CNS toxicity, disruption of Blood Brain barrier etc, like SAE, Dengue were observed. Post Covid Encephalopathy, Scrub typhus and Leptospira associated neurological involvement were also found among rare presentations. Outcome in patients with SAE with multiple organ involvement and shock was particularly adverse, TBM patients were found to recover well if the disease is not complicated by Hydrocephalus or large infarcts and need for neurosurgical intervention. Early diagnosis and prompt treatment in patients presenting with Acute Febrile Encephalopathy to the Emergency Department can impact the survival of these patients especially in the setting of a tertiary hospital care.

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S.NO	MRD NO	DOA	AGE	SEX	RELIGION	OCCUPATION	CHIEF COMPLAINTS FEVER	ALTERED SENSORIUM	HEADACHE	VERTIGO	BLURRING VISION	ABNORMAL BODY MOVEMENT	WEAKNESS IN ANY PART OF	VOMITING	OTHERS	PAST MEDICAL ILLNESS HISTORY			FAMILY HISTORY	PERSONAL HISTORY				SBP	DBP	TEMP	PULSE RATE	RR	PALOR	GCS	CLUBBING	ICTERUS	EDEMA	PETECHIAE / PURPURA
																TYPE 2 DM	HYPERTENSION	OTHERS		ALCOHOL	SMOKING	SUBSTANCE ABUSE	NONE										
1	874847	01-01-2020	90	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	Y	SLURRED SPEECH	-	Y	Y	NIL	-	-	-	Y	70	50	96.8	133	22	-	5	Y	-	-	-
2	862342	01-02-2020	67	M	SIKH	DEPENDENT	Y	Y	Y	N	N	N	N	Y	-	-	Y	-	NIL	-	-	-	Y	100	60	100	108	18	-	10	-	-	-	-
3	304741	01-02-2020	85	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	N	SHORTNESS OF BREATH	Y	Y	ILD	NIL	-	-	-	Y	100	70	98.7	122	36	-	11	-	-	-	-
4	561033	01-03-2020	82	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	PARKINSON'S	NIL	Y	-	-	-	110	70	98.2	120	20	-	12	-	-	-	-
5	545059	01-04-2020	69	M	HINDU	RETIRED	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	90	50	99.8	102	32	-	11	-	-	-	-
6	868479	01-04-2020	55	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	HYPOTHYROID	NIL	-	-	-	Y	90	60	100	92	18	Y	7	-	-	-	-
7	873240	01-05-2020	57	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	110	70	100	78	16	-	12	-	-	-	-
8	890003	01-06-2020	71	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	100	70	100	98	18	-	12	-	-	-	-
9	964110	10-1-20	24	F	SIKH	STUDENT	Y	Y	Y	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	80	50	102	120	22	Y	11	-	-	-	-
10	818975	10-1-20	67	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	100	60	103	122	16	-	9	-	-	-	-
11	304741	10-1-20	83	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	110	70	101	98	18	Y	12	Y	-	-	-
12	879385	11-1-20	78	M	SIKH	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	-	Y	-	NIL	Y	-	-	-	130	70	102	113	20	-	12	-	-	-	-
13	876754	13-01-2020	32	M	SIKH	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	PAIN ABDOMEN	-	-	-	NIL	-	-	-	Y	80	50	100	142	26	-	10	-	-	-	-
14	881058	14/1/20	26	M	SIKH	STUDENT	Y	Y	N	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	90	60	101	144	22	-	11	-	-	-	-
15	880949	14/1/20	76	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	SLURRING OF SPEECH	Y	-	PARKINSON'S	NIL	-	-	-	Y	90	40	99.3	145	20	-	6	-	-	-	-
16	290471	18/1/20	64	M	SIKH	RETIRED	Y	Y	N	N	N	N	N	N	-	-	-	OLD CVA	NIL	Y	-	-	-	100	60	99.8	108	22	-	12	-	-	-	-
17	884101	22/1/20	24	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	90	60	101	119	22	Y	10	-	-	-	-
18	863637	23/1/20	65	M	SIKH	DEPENDENT	Y	Y	N	Y	N	N	N	N	-	Y	-	-	NIL	Y	Y	-	-	100	70	100	99	18	-	12	-	-	-	-
19	884096	24/1/20	34	M	SIKH	FARMER	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	98.3	110	22	-	12	-	-	-	-
20	887170	25/1/20	86	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	SLURRING SPEECH	-	Y	-	NIL	-	-	-	Y	80	50	101	98	18	Y	11	-	-	-	-
21	884291	26/1/20	53	M	SIKH	FARMER	Y	Y	Y	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	120	60	99	108	20	-	12	-	-	-	-
22	884398	27/1/20	19	F	HINDU	STUDENT	Y	Y	Y	N	Y	N	N	N	-	-	-	-	TB IN FATHER	-	-	-	Y	110	70	101	121	16	-	12	-	-	-	-
23	888847	28/1/20	33	M	ISLAM	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	60	101	98	20	-	10	-	-	-	-
24	878127	29/1/20	55	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	Y	-	Y	-	-	NIL	-	-	-	Y	100	70	98.3	89	18	-	11	-	-	-	-
25	885296	1-2-20	55	F	SIKH	AGRICULTURE	Y	Y	N	N	Y	N	N	N	-	-	-	-	NIL	-	-	-	Y	90	60	97.3	90	17	-	12	-	-	-	-
26	875097	1-2-20	58	M	HINDU	SELF EMPLOYED	Y	Y	Y	N	N	N	N	N	-	N	-	-	NIL	-	-	-	Y	120	70	102	98	22	-	12	-	-	-	-
27	891293	2-2-20	61	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	DECREASED URINE OUTPUT	-	-	SLE	NIL	-	-	-	Y	100	70	99	102	18	-	10	-	-	-	-
28	892426	4-2-20	63	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	110	70	99.9	102	19	Y	11	-	-	-	-
29	889960	6-2-20	45	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	HYPOTHYROID	NIL	-	-	-	Y	60	40	100	96	21	-	10	-	-	-	-
30	889007	7-2-20	65	M	HINDU	LAWYER	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	99.6	67	15	-	11	-	-	-	-
31	891746	8-2-20	33	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	Y	-	-	-	NEUROFIBROMATOSIS	NIL	-	-	-	Y	110	70	100	123	17	-	4	-	-	-	-
32	895569	9-2-20	81	F	SIKH	DEPENDENT	Y	Y	Y	Y	N	Y	N	N	-	Y	-	-	NIL	-	-	-	Y	100	60	100	108	18	-	10	-	-	-	-
33	922067	11-2-20	69	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	SLURREING,FACE DEVIATION	Y	Y	-	NIL	-	-	-	Y	100	70	100	108	18	-	12	-	-	-	-
34	650529	12-2-20	75	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	DECREASED URINE OUPUT/DISCHARGE PER VAGINUM	Y	-	-	NIL	-	-	-	Y	80	60	100	110	18	-	10	-	-	-	-
35	898420	13/2/20	61	F	HINDU	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	90	60	101	89	17	Y	12	-	-	-	-
36	891980	14/2/20	51	M	SIKH	BUSINESS	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	Y	-	90	60	100	145	24	-	5	-	-	-	-
37	899897	16/2/20	27	M	HINDU	STUDENT	Y	Y	N	N	N	N	N	Y	SHORTNESS OF BREATH	-	-	ACYANOTIC HEART DISEASE	NIL	-	-	-	Y	110	70	102	122	18	-	12	-	-	-	-
38	901499	21/2/20	51	M	HINDU	BUSINESS	Y	Y	N	N	N	Y	N	N	-	Y	-	-	NIL	-	-	-	Y	110	70	99.4	88	18	-	10	-	-	-	-
39	904082	22/2/20	82	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	100	60	98.3	110	22	-	11	-	-	-	-
40	897781	24/2/20	43	M	HINDU	ENGINEER	Y	Y	N	N	N	N	N	N	INTUBATED OUTSIDE	-	-	-	NIL	Y	-	-	-	90	60	100	134	28	-	3	-	-	-	-
41	905063	24/2/20	77	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	Y	H/O FALL	Y	Y	-	NIL	-	-	-	Y	100	60	99.8	108	22	-	11	-	-	-	-
42	905854	25/2/20	62	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	LOSS OF APETITE	-	Y	OA	NIL	-	-	-	Y	90	50	100	121	19	-	12	-	-	-	-
43	899846	25/2/20	58	F	HINDU	DEPENDENT	Y	Y	Y	N	Y	Y	N	N	PTOSIS	-	-	OA KNEE	NIL	-	-	-	Y	100	70	97	90	13	Y	12	-	-	-	-

S.NO	MRD NO	LYMPHADENOPATHY	CYANOSIS	CENTRAL NERVOUS SYSTEM	CVS	RS	P/A	HB	TLC	DLC	PLT	ESR	PBF MALARIA	PTI-INR	UREA	CREATININE	NA	K	S. BILIRUBIN	TOTAL PROTEIN	ALBUMIN	ALP	SGOT	SGPT	CALCIUM	ABG	CXR	ECG	BLOOD C/S/URINE C/S	Malaria Antigen
1	874847	-	-	HMF ALTERED,B/L PLANATR MUTE,MOVING ALL 4 LIMBS	NAD	NAD	NAD	10.9	16.1	88/6/8	295	33	NEG	1.18	89	1.69	130	5	0.3	7.2	3.4	131	28	32	7.8	ACIDOSIS	NORMAL	NORMAL	1	NA
2	862342	-	-	HMF ALTERED, B/LPLANTAR EXTENSOR	NAD	NAD	NAD	9.9	10.1	72/18/10	226	90	NEG	0.98	67	1.55	143	3.6	3.44	7.1	3.4	161	319	242	8	NORMAL	NORMAL	NORMAL	1	NEG
3	304741	-	-	HMF ALTERED	ABNORMAL	B/L CREPTS PRESENT	NAD	8.4	9.9	92/4/3	38	65	NEG	1.78	32	1.19	138	4.15	0.71	5.7	2	151	29	23	8	TYPE 1 RF	B/L DIFFUSE INFILTERATES	NORMAL	1	NEG
4	561033	-	-	HMF ALTERED	NAD	B/LCREPTS PRESENT	NAD	9.8	14	76/15/7	263	40	NEG	1.22	126	4.5	145	4.6	1.35	7.8	2.66	384	277	169	8.6	ACIDOSIS	B/L DIFFUSE INFILTERATES	ABNORMAL	URINEC/S: E.COLI	NEG
5	545059	-	-	HMF ALTERED	NAD	B/L CREPTS PRESENT	NAD	8	6.7	76/14/6/4	130	87	NEG	1.09	73	3.98	143	6	0.89	7.6	2.47	210	24	15	8.4	ACIDOSIS	B/L LZ HAZINESS	ABNORMAL	1	NEG
6	868479	-	-	DROWSY,DISORIENTED,POWER PRESERVED	NAD	NAD	NAD	12.7	6	87/8	159	65	NEG	1.17	24	0.4	131	3.6	1.08	5.6	2.8	160	45	54	7.9	NORMAL	NORMAL	NORMAL	1	NEG
7	873240	-	-	HMF ALTERED	NAD	NAD	NAD	11.6	9.9	82/10/8	120	45	NEG	1.23	24	0.52	127	4.5	0.47	6	3.43	53	41	24	8.8	NORMAL	NORMAL	NORMAL	1	NEG
8	890003	-	-	HMF ALTERED	NAD	NAD	NAD	11	8.8	68/9/3	216	82	NEG	1.23	30	0.3	128	3.6	0.8	7	3.6	88	42	38	8.9	NORMAL	MILIARY SHADOWS	NORMAL	1	NEG
9	964110	Y	-	CONFUSED,NO VERBAL RESPONSE	NAD	NAD	NAD	10.6	9.2	82/15/6	335	77	NEG	1.2	40	0.34	132	4	0.57	7	4	113	41	18	7.2	NORMAL	NORMAL	NORMAL	1	NEG
10	818975	-	-	HMF ALTERED, B/L PLANATAR UPGOING,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	11	13	88/10/2	314	16	NEG	1.34	30	0.5	133	4	0.8	6.3	2.83	98	65	71	8.9	NORMAL	NORMAL	NORMAL	1	NEG
11	304741	-	-	HMF ALTERED	NAD	B/L AE REDUCED	NAD	6	5.7	45/55	60	34	NEG	1.45	87	2.3	135	4.3	0.8	5.6	2.45	89	1034	760	7	LACTIC ACIDOSIS	NORMAL	NORMAL	1	NEG
12	879385	-	-	HMF ALTERED,MENINGEAL SIGNS	NAD	NAD	NAD	13.3	29	92/8	177	35	NEG	1.28	32	0.78	133	3.3	1.77	7.9	3.67	95	23	34	8	NORMAL	NORMAL	NORMAL	SPUTUM:KLEBSIELLA	NEG
13	876754	-	-	HMF ALTERED, MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	15.4	10.9	74/20/5	15	35	NEG	1.19	21	0.92	143	3.76	1.95	4.9	2.24	228	679	512	8.7	ACIDOSIS	B/L LOWER ZONES INFILTRATES	NORMAL	1	NEG
14	881058	-	-	HMF ALTERED,NO MENINGEALSIGNS	NAD	B/L REDUCED AIR ENTRY	NAD	8.4	30	87/7/4	139	100	NEG	1.19	31	0.75	139	4.6	2.17	5.9	2.87	139	405	719	8	ACIDOSIS	B/L PLEURAL EFFUSION	ABNORMAL	1	NEG
15	880949	-	-	HMF ALTERED,RIGHT HEMIPARESIS	NAD	B/L DIFFUSE CONDUCTED SOUNDS	NAD	9.6	9.7	70/22/8	214	88	NEG	0.97	72	1.18	131	6	0.46	5.6	2.74	75	20	25	8.1	ACIDOSIS	B/L DIFFUSE INFILTERATES	ABNORMAL	1	NEG
16	290471	-	-	HMF ALTERED	NAD	NAD	RIGHT SIDED FECOLITHS	11.6	6	62/21/8	150	88	NEG	1.32	24	0.54	130	3.7	0.4	7	4.1	88	43	34	9	NORMAL	NORMAL	NORMAL	URINE C/S: E.COLI	NEG
17	884101	-	-	HMF ALTERED,MENINGEAL SIGNS	NAD	NAD	NAD	8.8	2.8	85/10/5	198	35	NEG	1.12	10	0.34	132	4.15	0.08	5.6	2.99	100	66	60	9	METABOLIC ACIDOSIS	NORMAL	NORMAL	1	NEG
18	863637	-	-	HMF ALTERED	NAD	B/L LZ CREPTS	HEPATOMEGALY	9.9	17.2	84/10/6	617	100	NEG	1.19	94	1.38	145	4.5	0.65	6.1	2.34	63	33	50	8.5	ACIDOSIS	B/L LZ HAZINESS	NORMAL	SPUTUM C/S:KLEBSIELLA/ WOUND SWAB: MRSA	NEG
19	884096	Y	-	DROWSY,CONFUSED,DIORIENTED	NAD	NAD	NAD	9.9	4.6	78/20/2	190	32	NEG	1.23	31	0.23	136	4	0.29	6.5	3.88	94	22	61	8	NORMAL	NORMAL	NORMAL	1	NEG
20	887170	-	-	HMF ALTERED	NAD	NAD	NAD	7.7	16.8	89/10/1	174	56	NEG	1	91	1.31	140	4.9	0.16	6.8	3.48	104	111	154	9.5	ACIDOSIS	NORMAL	ABNORMAL	1	NEG
21	884291	-	-	CONFUSED,RESTLESS,NUCHAL RIGIDITY,DECREASED POWER IN B/L LL,INVOLUNTARY JERKS IN LEFT UL	NAD	NAD	NAD	10.2	13.6	75/18/6	193	45	NEG	1.1	14	0.48	122	3.6	0.24	5.8	2.94	89	57	63	8.4	NORMAL	NORMAL	NORMAL	URINE C/S CANDIDA	NEG
22	884398	Y	-	IRRITABLE,DISORIENTED,	NAD	NAD	NAD	12	14.8	80/15/5	169	63	NEG	1.64	22	0/6	135	3.9	O.58	7	3.6	78	9	13	8.2	NORMAL	NORMAL	NORMAL	1	NEG
23	888847	-	-	HMF ALTERED	NAD	NAD	NAD	13.9	10	68/21/10	116	22	NEG	1.74	165	5.83	140	4.3	1.15	6.1	2.11	122	166	178	9	NORMAL	B/L DIFFUSE INFILTERATES	NORMAL	1	NEG
24	878127	-	-	HMF ALTERED	NAD	NAD	NAD	11	18	76/18/8	167	19	NEG	1.25	20	0.5	145	3.2	0.9	7	3.4	170	78	67	7.3	NORMAL	NORMAL	NORMAL	1	NEG
25	885296	Y	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	7.6	11.2	82/16/2	234	45	NA	1.25	40	0.5	134	4.2	0.6	6.1	2.8	88	45	90	9	NORMAL	NORMAL	NORMAL	1	NEG
26	875097	-	-	HMF ALTERED	NAD	NAD	NAD	15.2	10.3	76/20/4	188	35	NEG	1.29	28	0.93	136	4.5	0.49	6.9	4.04	57	26	94	8	RESPIRATORY ALKLOSIS	NORMAL	ABNORMAL	1	NEG
27	891293	-	-	HMF ALTERED	NAD	NAD	NAD	9.5	8.8	71/15/10	120	24	NEG	1.12	73	6.22	139	3.5	0.58	5.5	2.81	180	34	13	7.8	ACIDOSIS	LEFT MIDDLE ZONE PATCH	NORMAL	1	NEG
28	892426	-	-	HMF ALTERED	NAD	B/L CREPTS	NAD	10.6	17.1	84/8/6/2	120	46	NEG	1.26	55	2.12	135	3.8	0.51	5.9	3.16	76	59	43	10	ACIDOSIS	NORMAL	ABNORMAL	URINE C/S: E.COLI	NEG
29	889960	-	-	DROWSY,NO VERBAL RESPONSE,WITHDRWL ON PAIN	NAD	NAD	NAD	9	22	73/20/6/0	175	82	NEG	1.13	32	0.56	138	3.9	0.83	7	3	458	448	110	8.6	NORMAL	NORMAL	NORMAL	1	NEG
30	889007	Y	-	CONFUSED,MOVING ALL LIMBS,NOT FOLLOWING COMMANDS,ROWSY	NAD	NAD	NAD	9.2	11.2	82/8/2	155	73	NEG	1.28	58	0.96	140	3.9	0.63	7	3.4	62	32	46	8.2	NORMAL	NORMAL	NORMAL	1	NEG
31	891746	-	-	INTUBATED,B/L PLANTAR MUTE,RIGHT HEMIPARESIS,MENGEAL SIGNS PRESENT	NAD	NAD	NAD	11	15.2	92/6/3	234	36	NA	1.15	34	0.47	138	4.3	0.71	5.7	3.04	108	24	33	7.2	RESPIRATORY ALKALOSIS	B/L LZ HAZINESS	ABNORMAL	1	NEG
32	895569	-	-	HMF ALTERED	NAD	NAD	NAD	10.2	13.4	85/10/5	178	54	NA	1.21	106	5.6	139	5.8	0.45	5.3	2.31	156	99	78	8.1	ACIDOSIS	NORMAL	NORMAL	BLOOD C/S: E.COLI	NEG
33	922067	-	-	SLURRED SPEECH/FACIAL DEVIATION	NAD	NAD	NAD	7.8	11	76/14/10	260	16	NEG	1.19	28	0.89	140	3.9	0.61	6.7	3.8	66	135	225	8	NORMAL	NORMAL	NORMAL	1	NEG
34	650529	-	-	HMF ALTERED	ABNORMAL	B/L CREPTS	NAD	8.1	14.3	89/9/1	156	45	NEG	1	129	2.93	129	5	2.1	4.8	2	125	87	98	7.1	ACIDOSIS	NORMAL	ABNORMAL	1	NEG
35	898420	-	-	HMF ALTERED, MENINGEAL SIGNS PRESENT	NAD	B/L SPASM PRESENT	NAD	8	6.1	68/18/8	202	56	NEG	1.32	10	0.6	145	3.56	4	5.9	2.63	94	31	24	9	NORMAL	EMPHYSEMATOUS CHANGES	NORMAL	1	NA
36	891980	-	-	HMF ALTEERED,MOVING ALL 4 LIMBS	NAD	DECREASED AIR ENTRY ON RIGHT SIDE	NAD	10.6	15.7	90/8/2	145	12	NA	1	145	3.58	150	3.5	0.34	5.3	2.65	240	47	49	6.8	ACIDOSIS	RIGHT PLEURALEFFUSION	NORMAL	1	NA
37	899897	-	-	HMF ALTERED	ABNORMAL	B/L FINE CREPTS	NAD	11.7	7.8	85/8/5	40	45	NEG	1.12	28	1.95	135	3.7	0.77	5.9	3.13	143	59	42	8	NORMAL	B/LLZ HAZINESS	ABNORMAL	1	NEG
38	901499	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	10.2	11.3	85/8/5	162	19	NEG	1.2	43	0.6	129	3.8	1.86	0.9	3.2	90	87	78	9	NORMAL	NORMAL	ABNORMAL	1	NEG
39	904082	-	-	HMF ALTERED	NAD	NAD	NAD	6.8	16	88/10/2	537	34	NEG	1.19	25	0.59	133	4.6	0.31	5.5	2.21	130	26	20	8.3	NORMAL	NORMAL	NORMAL	URINE/BLOOD C/S: KLEBSIELLA	NEG
40	897781	-	-	MENINGEAL SINGS PRESENT,TONE WAS INCREASED OVERALL	ABNORMAL	B/L CREPTS	NAD	12.3	21.2	90/2.5/7.3/0	346	18	NEG	1.18	30	1.01	134	3.5	0.59	6.2	3.2	104	85	45	8.2	METABOLIC ACIOSIS	FLUID OVERLOAD	ABNORMAL	BLOOD C/S: CANDIDA	NEG
41	905063	-	-	HMF ALTERED	NAD	B/L DECREASED AIR ENTRY	NAD	9.4	14.6	88/10/2	207	26	NEG	1.21	112	3.77	133	4.75	0.98	5.5	2.57	149	35	14	7.1	ACIDOSIS	B/L PLEURAL EFFUSION	NORMAL	URINE C/S: KLEBSIELLA	NEG
42	905854	-	-	HMF ALTERED	NAD	RIGHT BRONCHIAL BREATHING/CREPTS	NAD	10	23.7	84/10/6	637	21	NEG	1.33	130	2.99	136	3.73	0.36	6.8	3.58	66	14	9	7	ACIDOSIS	RIGHT SIDED PNEUMONITIS	NORMAL	1	NEG
43	899846	-	-	DROWSY,RT 3RD N PALSY,MOVING ALL 4 LIMBS	NAD	NAD	NAD	14	6.8	82/20/5/0	237	114	NEG	1.13	24	0.38	133	4.16	1.48	4.7	2.2	127	18	64	7.8	NORMAL	NORMAL	NORMAL	1	NEG

S.NO	MRD NO	Leptospira	Dengue	Scrub Typhus	Other Investigations	CSF CYTOLOGY_TLC	NEUTROPHILS	LYMPHOCYTES	GLUCOSE	S. GLUCOSE	PROTEIN	CHLORIDE	GRAM STAIN	INDIA INK PREPARATION	BACTERIAL ANTIGEN	CULTURE SENSITIVITY	ADA	CBNAAT	HSV PCR	CT HEAD_FOCAL HYPODENSITIES	CT HEAD HYDROCEPHALUS	CT HEAD_DIFFUSE CEREBRAL OEDEMA	CT HEAD INFARCT	CT HEAD OTHERS	CT HEAD NORMAL	MRI LEPTOMENINGITIS	MRI VASCULITIC INFARCT
1	874847	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
2	862342	NA	NA	NA	-	2	0	0	52	102	87	109	-	-	-	-	10.5	-	-	N	Y	N	N	N	N	N	N
3	304741	NEG	NEG	NEG	ANTI CCP441,ANA:4+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
4	561033	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
5	545059	NEG	NEG	NEG	GALACTOMANNAN: 2.89	2	0	0	102	117	34	120	-	-	-	-	1.34	-	-	N	N	N	N	N	Y	N	N
6	868479	NA	NA	NA	-	30	20	80	38	172	120	107	NAD	NAD	NEG	NIL	20.55	-	-	Y	Y	N	Y	N	N	Y	Y
7	873240	NA	NA	NA	-	10	10	90	70	104	32	95	-	-	-	-	35	-	-	N	N	N	N	N	Y	N	N
8	890003	NA	NA	NA	-	80	20	80	26	128	206	102	-	-	-	-	35	-	-	N	Y	N	N	N	N	Y	N
9	964110	NA	NA	NA	-	40	0	100	18	124	359	104	-	-	-	-	13	-	-	N	Y	N	N	N	N	N	N
10	818975	NA	NA	NA	-	2	10	90	102	110	147	116	-	-	-	-	2	-	POSITIVE	N	N	N	N	N	N	N	N
11	304741	NEG	POSITIVE	NEG	FUNGITELL 272	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
12	879385	NA	NA	NA	-	110	10	90	50	89	563	113	-	-	POSITIVE	-	14	-	-	N	N	N	N	N	N	N	N
13	876754	NEG	NEG	POSITIVE	USG :HEPTOSPLENOMEGALY	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
14	881058	NEG	NEG	NEG	PCT 28	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
15	880949	NEG	NEG	NEG	PCT 22	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
16	290471	NEG	NEG	NEG	PCT 7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
17	884101	NA	NA	NA	-	20	0	100	50	98	80	105	-	-	-	-	16.1	POSITIVE	-	N	Y	N	N	N	N	Y	N
18	863637	NA	NA	NA	SEPTIC ARTHRITIS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
19	884096	NA	NA	NA	-	13	5	95	65	110	58	70	-	-	-	-	75	-	-	N	N	N	N	N	Y	Y	N
20	887170	NA	NA	NA	PCT 0.5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
21	884291	NA	NA	NA	-	10	0	100	38	108	236	113	-	-	-	-	16	-	-	N	N	N	N	N	N	Y	N
22	884398	NA	NA	NA	-	500	20	80	58	165	56	123	NAD	NAD	NEG	NIL	15	-	-	N	N	N	N	N	Y	Y	N
23	888847	NEG	NEG	POSITIVE	PCT 0.09	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
24	878127	NA	NA	NA	-	1500	95	5	53	167	190	112	-	-	-	-	23	-	-	N	N	N	N	N	N	Y	N
25	885296	NA	NA	NA	-	100	10	90	54	109	289	110	-	-	-	-	62	-	-	Y	N	N	N	N	N	Y	N
26	875097	NA	NA	NA	-	300	10	90	37	132	144	113	-	POSITIVE	-	-	16	-	-	N	N	N	N	N	N	Y	Y
27	891293	NEG	NEG	NEG	PCT 3.9	5	0	0	89	102	63	109	-	-	-	-	8.5	-	-	N	N	N	N	N	Y	N	N
28	892426	NEG	NEG	NEG	PCT 35	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
29	889960	NA	NA	NA	-	20	65	35	68	142	117	127	-	-	-	-	2	-	POSITIVE	N	N	N	N	N	N	N	N
30	889007	NA	NA	NA	-	30	10	90	98	261	58	119	-	-	-	-	11.5	-	-	N	N	N	N	N	N	Y	N
31	891746	NA	NA	NA	-	1200	70	30	78	99	610	148	-	-	STREPTOCOCCUS PNEUMONIAE	-	49	-	-	N	N	N	N	N	N	Y	Y
32	895569	NA	NA	NA	PCT 60	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
33	922067	NA	NA	NA	-	15	80	20	65	121	67	108	-	-	-	-	12	-	-	N	N	N	N	N	Y	N	Y
34	650529	NA	NA	NA	PCT 8, PYOMETRA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
35	898420	NA	NA	NA	-	2	0	0	70	110	35.2	122	-	-	-	-	1.4	-	NEGATIVE	N	N	N	N	N	N	N	N
36	891980	NA	NA	NA	PCT 14	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
37	899897	POSITIVE	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
38	901499	NA	NA	NA	-	200	10	90	87	184	89	111	-	-	-	-	9	-	POSITIVE	N	N	N	N	N	N	N	N
39	904082	NEG	NEG	NEG	PCT 28,NCCT KUB : GROSS HUN	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
40	897781	NEG	NEG	NEG	2D ECHO-LVEF 22 GHK	20	20	80	98	110	99	108	-	-	-	-	-	-	-	N	N	Y	N	N	N	N	N
41	905063	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
42	905854	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
43	899846	NA	NA	NA	-	252	4	96	62	170	308	-	NO GROWTH	-	-	-	15	-	-	N	N	N	N	N	N	Y	N

S.NO	MRD NO	MRI_RING ENHANCING LESIONS	MRI_MENINGIOENCEPHALITIS/ ENCEPHALITIS	MRI HYDROCEPHALUS	MRI_DIFFUSE CERBRAL OEDEMA	MRI NORMAL	MRI_OTHERS	EEG (if needed)	TUBERCULOUS MENINGITIS	2.VIRAL	3.DENGUE ENCEPHALOPATHY/ ENCEPHALITIS	4.PYOGENIC	5.MALARIA	6.SCRUB RELATED ENCEPHALOPATHY	7.SEPTIC ENCEPHALOPATHY	POST COVID ENCEPHALOPATHY	LEPTOSPIRA INDUCED ENCEPHALOPATHY	OTHERS	TREATMENT GIVEN	OUTCOME 1. RECOVERED, 2. DAMA,
1	874847	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ANTIVIRALS	1
2	862342	N	N	Y	N	N	HYPOXIC INJURY	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/VENTILATOR	3
3	304741	N	N	N	N	N	-	-	N	N	N	N	N	N	N	N	N	N	IV ANTIVIOTICS/ANTIFUNGALS/STEROIDS/INOTROPES/VENTILATOR	3
4	561033	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
5	545059	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/INOTROPES/SUPPORTIVE	2
6	868479	Y	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/IV ANTIBIOTICS	2
7	873240	N	N	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIBIOTICS/ANTIVIRAL/ANTIEPILEPTICS	1
8	890003	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
9	964110	N	Y	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
10	818975	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	ANTIVIRALS/ANTIEPILEPTICS	1
11	304741	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS/ANTIFUNGALS,VENTILATORY SUPPORT	3
12	879385	N	N	N	N	N	-	-	N	N	N	STREPTOCOCCUS PNEUMONIAE	N	N	N	N	N	N	IV ANTIBIOTICS,STEROIDS	1
13	876754	N	N	N	N	N	-	-	N	N	N	N	N	Y	N	N	N	N	IV DOXYCYCLINE, IB MERPENEM,INOTROPES,VENTILATORY SUPPORT,IV NAC	3
14	881058	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS	1
15	880949	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ANTIPLATLET/FLUIDS	1
16	290471	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
17	884101	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
18	863637	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ARTHROTOMY/SUPPORTIVE	1
19	884096	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
20	887170	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	2
21	884291	N	Y	N	N	N	ARACHNOIDITIS	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
22	884398	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
23	888847	N	N	N	N	N	-	-	N	N	N	N	N	N	N	N	Y	N	IV MONOCEF/IV ANTIBIOTICS	2
24	878127	N	N	N	N	N	-	-	N	N	N	Y	N	N	N	N	N	N	IV ANTIBIOTICS/STEROIDS	1
25	885296	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
26	875097	N	N	N	N	N	-	-	N	N	N	N	N	N	N	N	N	CRYPTOCOCCALMENINGITIS	LIPOSOMAL AMPHOTERICIN B/FLUCONAZOLE	1
27	891293	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/	1
28	892426	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	2
29	889960	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	ANTIVIRAL/STEROIDS	2
30	889007	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/NEKIUM INFUSION	2
31	891746	N	N	N	N	N	VENTRICULITIS	-	N	N	N	STREPTOCOCCUS PNEUMONIAE	N	N	N	N	N	N	IV ANTIBIOTICS,IV STEROIDS	2
32	895569	N	N	N	Y	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV NATIVIOTICS/STEROIDS/INOTROPES1	1
33	922067	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/ANTIPLATELET DRUGS	1
34	650529	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/INOTROPES/DIAALYSIS	3
35	898420	N	N	N	N	N	DIFFUSE CEREBRAL ATROPHY	-	N	Y	N	N	N	N	N	N	N	N	ANTI VIRAL/ANTIEPILEPTIC/STEROID/IV ANTIBIOTICS	1
36	891980	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS,VENTILATOR,INOTROPES	3
37	899897	N	N	N	N	Y	-	-	N	N	N	N	N	N	N	N	Y	N	IV MONOCEF/AZEE	1
38	901499	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	ANTIVIRAL/ANTIEPILEPTIC/STEROIDS	1
39	904082	N	N	Y	N	N	CHRONIC HYPERTENSIVE MICROANGIOPATHY	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ANTIEPILEPTICS	1
40	897781	N	N	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRALS/ANTIBIOTICS	1
41	905063	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ SUPPORTIVE	1
42	905854	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
43	899846	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROID/ANTI EPI	1

S.NO	MRD NO	DOA	AGE	SEX	RELIGION	OCCUPATION	CHIEF COMPLAINTS FEVER	ALTERED SENSORIUM	HEADACHE	VERTIGO	BLURRING VISION	ABNORMAL BODY MOVEMENT	WEAKNESS IN ANY PART OF	VOMITING	OTHERS	PAST MEDICAL ILLNESS HISTORY			FAMILY HISTORY	PERSONAL HISTORY				SBP	DBP	TEMP	PULSE RATE	RR	PALOR	GCS	CLUBBING	ICTERUS	EDEMA	PETECHIAE / PURPURA
																TYPE 2 DM	HYPERTENSION	OTHERS		ALCOHOL	SMOKING	SUBSTANCE ABUSE	NONE										
44	906511	26/2/20	70	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	DECREASED URINE OUTPUT,SHORTNESS OF BREATH	Y	Y	OLD CVA	TYPE 2 DM IN MOTHER AND SON	-	-	-	Y	100	70	99.8	89	19	-	11	-	-	-	-
45	905849	27/2/20	26	M	HINDU	SERVICE	Y	Y	Y	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	100	70	102	108	22	Y	10	-	-	-	-
46	907879	28/2/20	63	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	Y	-	NIL	-	-	-	Y	110	70	101	121	18	-	11	-	-	-	-
47	869413	28/2/20	50	M	SIKH	FARMER	Y	Y	Y	N	N	N	N	N	-	-	Y	-	NIL	Y	-	-	-	140	90	98	72	14	-	10	-	-	-	-
48	903037	1-3-20	29	M	SIKH	BUSINESS	Y	Y	N	N	N	Y	N	N	-	Y	-	-	NIL	Y	-	-	-	110	70	98.3	88	17	-	12	-	-	-	-
49	909322	2-3-20	73	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	Y	-	Y	Y	-	NIL	-	-	-	Y	80	40	98.5	98	19	-	10	-	-	-	-
50	899137	2-3-20	66	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	HYPOTHYROID	NIL	-	-	-	Y	100	60	100	87	16	-	12	-	-	-	-
51	904335	3-3-20	76	M	MUSLIM	SERVICE	Y	Y	N	N	N	N	N	N	B/L LOWER LIMB WEAKNESS	-	Y	-	NIL	-	-	-	Y	110	70	101	108	16	-	11	-	-	-	-
52	792833	3-3-20	32	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	120	70	99.2	89	16	-	12	-	-	-	-
53	908748	3-3-20	29	F	HINDU	DEPENDENT	Y	Y	N	N	Y	N	N	N	-	-	-	HYPOTHYROID	NIL	-	-	-	Y	100	60	100	88	15	-	12	-	-	-	-
54	910213	3-3-20	61	M	HINDU	RETIRED	Y	Y	N	N	N	N	N	N	SHORTNESS OF BREATH	Y	-	-	NIL	-	-	-	Y	90	60	99.4	108	28	Y	7	-	-	-	-
55	910995	4//3/20	65	M	HINDU	RETIRED	Y	Y	N	N	N	N	N	N	-	-	-	CA LOWER ESOPHAGUS	NIL	-	-	-	Y	90	60	101	103	18	Y	12	-	-	-	-
56	908077	4-3-20	28	M	HINDU	Business	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	99	92	15	-	15	-	-	-	-
57	909520	6-3-20	75	M	HINDU	SELF EMPLOYED	Y	Y	N	N	N	Y	N	N	-	-	-	-	H/O TB IN PAST	-	-	-	Y	100	60	100	98	16	-	10	Y	-	-	-
58	906529	7-3-20	58	M	SIKH	CARPENTER	Y	Y	N	N	N	N	N	N	-	-	-	HIV	NIL	-	-	-	Y	100	70	98	87	22	-	12	-	-	-	-
59	907966	8-3-20	22	F	HINDU	STUDENT	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	120	60	100	119	25	Y	5	-	-	-	-
60	912943	9-3-20	67	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	-	Y	HYPOTHYROID	NIL	-	-	-	Y	140	90	99	88	20	Y	12	-	-	-	-
61	906464	10-3-20	54	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	-	Y	Y	-	NIL	-	-	-	Y	100	70	100	110	20	-	11	-	-	Y	-
62	916640	15/3/20	72	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	Y	-	NIL	-	-	-	Y	110	70	99.8	108	19	-	9	-	-	-	-
63	916165	15/3/20	32	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	120	60	102	88	18	-	12	-	-	-	-
64	606618	16/3/20	28	F	HINDU	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	-	-	SOLITARY RIGHT KIDNEY	NIL	-	-	-	Y	90	60	99	128	22	-	6	-	-	-	-
65	885296	16/3/20	55	F	SIKH	DEPENDENT	Y	Y	Y	N	N	N	Y	N	-	-	-	-	NIL	-	-	-	Y	130	70	101	125	15	-	12	-	-	-	-
66	912359	16/3/20	18	M	HINDU	STUDENT	Y	Y	Y	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	110	70	102	132	16	-	11	-	-	-	-
67	919231	19/3/20	65	M	SIKH	SERVICE	Y	Y	N	N	N	N	N	N	INCREASED MICTURATION FREQUENCY	-	-	-	NIL	Y	-	-	-	80	60	103	167	28	-	10	-	-	-	-
68	919663	20/3/20	72	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	Y	-	NIL	-	-	-	Y	100	70	100	120	18	-	12	-	-	-	-
69	918746	20/3/20	81	F	SIKH	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	100	70	105	133	21	-	11	-	-	-	-
70	909443	20/3/20	53	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	80	100	85	20	-	11	-	-	-	-
71	919527	21/3/20	75	M	HINDU	RETIRED	Y	Y	N	N	N	N	N	N	-	Y	-	PARKINSON'S	NIL	-	-	-	Y	100	70	99.8	98	18	-	10	Y	-	-	-
72	920169	25/3/20	32	F	HINDU	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	100	60	102	135	26	-	10	-	-	-	-
73	916538	20/3/20	30	M	SIKH	BUSINESS	Y	Y	N	N	N	Y	N	N	-	-	-	-	HEMATOLOGICAL MALIGNANCY IN FATHER	-	-	-	Y	100	70	99.6	96	16	-	12	-	-	-	-
74	394431	23/3/20	44	F	HINDU	DEPENDENT	Y	Y	Y	N	N	Y	N	Y	-	-	Y	-	NIL	-	-	-	Y	140	70	103	121	26	-	10	-	-	-	-
75	887278	28/3/20	66	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	100	60	98	64	21	-	4	-	-	-	-
76	919951	1-4-20	61	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	Y	-	-	Y	-	NIL	-	-	-	Y	110	80	100	86	20	-	11	-	-	-	-
77	919674	11-4-20	73	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	-	-	Y	OLD CVA	NIL	Y	-	-	-	100	50	98.3	108	18	-	11	-	-	-	-
78	920784	11-4-20	48	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	-	Y	-	-	H/O TB IN FATHER	-	-	-	Y	120	80	98.6	86	15	-	12	-	-	-	-
79	774837	13/4/20	58	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	Y	DECREASED URINEOUTPUT	Y	Y	RA	NIL	-	-	-	Y	110	70	99.8	114	22	-	7	-	-	-	-
80	933611	16/4/20	64	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	RIGHT DIABTIC FOOT,PYURIA	Y	-	CAD	NIL	Y	-	-	-	100	60	99	110	20	-	10	-	-	-	-
81	920626	21/4/20	18	M	SIKH	STUDENT	Y	Y	Y	N	N	Y	N	Y	-	-	-	-	NAD	-	-	-	Y	100	60	102	98	22	-	10	-	-	-	-
82	760453	21/4/20	69	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	CA RECTUM	NIL	-	-	-	Y	90	60	99.6	108	18	-	12	-	-	-	-
83	922167	23/4/20	76	M	HINDU	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	-	-	-	MILIARY TB	TB IN YOUNGER SON	-	-	-	Y	100	70	101	110	17	-	12	Y	-	-	-
84	923527	24/4/20	60	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	DECREASED APETITE	Y	-	-	NIL	-	-	-	Y	90	60	100	128	18	-	11	-	-	-	-
85	923849	26/4/20	62	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	Y	-	NIL	-	-	-	Y	90	60	101	122	18	-	11	-	-	-	-

S.NO	MRD NO	LYMPHADENOPATHY	CYANOSIS	CENTRAL NERVOUS SYSTEM	CVS	RS	P/A	HB	TLC	DLC	PLT	ESR	PBF MALARIA	PTI-INR	UREA	CREATININE	NA	K	S. BILIRUBIN	TOTAL PROTEIN	ALBUMIN	ALP	SGOT	SGPT	CALCIUM	ABG	CXR	ECG	BLOOD C/S/URINE C/S	Malaria Antigen
44	906511	-	-	HMF ALTERED	ABNORMAL	B/L CREPTS PRESENT	NAD	12.5	18.4	95/4/1	45	41	NEG	1.39	94	3.86	135	5.1	0.67	5.9	3.01	151	76	39	8.1	ACIDOSIS	RIGHT LZ HAZINESS	ABNORMAL	URINE C/S: E.COLI	NEG
45	905849	-	-	HMF ALTERED, MENINGEALSINGS PRESENT	NAD	NAD	NAD	8.6	11.4	70/25/5	189	34	NEG	1.23	10	0.6	138	4.2	0.87	6.2	3.14	89	67	45	9.2	NORMAL	B/LINCREASED BRONCHOVASCULAR MARKINGS	NORMAL	1	NEG
46	907879	-	-	HMF ALTERED	NAD	NAD	NAD	6.1	9.4	90/8/2	303	22	NEG	1.21	197	12.3	144	6	0.36	5.6	2.47	132	69	43	8.9	NORMAL	NORMAL	NORMAL	1	NEG
47	869413	-	-	LEFT SIDED CEREBELLAR SIGNS PRESENT	NAD	NAD	NAD	15	9.2	77/15/5/1	289	85	NEG	1.58	32	0.6	135	3.6	0.6	7.3	2.63	88	88	60	10	NORMAL	NORMAL	NORMAL	1	NEG
48	903037	-	-	HMF ALTERED	NAD	NAD	NAD	11.2	7.8	52/28/2/1	238	7	NEG	1.28	30	0.7	143	3.8	0.73	7.5	4.5	71	60	42	8.1	NORMAL	NORMAL	NORMAL	1	NEG
49	909322	-	-	HMF ALTERED, NECK RIGIDITY,B/LPLANTAR UPGOING	ABNORMAL	B/L DECREASED AIR ENTRY	NAD	11	10.8	83/8/2	179	34	NEG	1.03	8	0.47	117	3.5	0.56	6.5	3.45	99	39	27	8.2	LACTIC ACIDOSIS	B/L PLEURAL EFFUSION	ABNORMAL	1	NA
50	899137	-	-	HMF ALTERED,NUCHAL RIGIDITY PRESENT	NAD	NAD	NAD	11.5	11.5	93/5/2	160	54	NA	1.44	50	0.7	124	4	0.9	6.7	3.4	98	25	65	8	NORMAL	MILIARY MOTTLING	NORMAL	1	NEG
51	904335	-	-	HMF ALTERED	NAD	NAD	NAD	11.5	8.6	86/7/5	135	90	NEG	1.41	26	0.51	130	3.09	0.99	6.5	2.73	61	15	13	9	NORMAL	NORMAL	ABNORMAL	1	NEG
52	792833	-	-	DROWSY,DISORIENTED,RESTLESS	NAD	NAD	NAD	10.3	11.3	76/23/5	134	66	NEG	1.27	34	0.8	132	4	0.9	6.3	4.2	88	12	98	9	NORMAL	NORMAL	NORMAL	1	NEG
53	908748	Y	-	HMF ALTERED	NAD	NAD	NAD	10.7	4.7	68/22/8	156	78	NA	1.34	33	0.2	137	4	0.8	6.9	3.3	108	45	78	7	NORMAL	NORMAL	NORMAL	1	NA
54	910213	-	-	HMF ALTERED	NAD	B/L DIFFUSE CREPTS	UMBILICAL HERNIA	7.9	12.6	83/12/5	140	32	NEG	1.45	165	5.02	143	3.8	0.45	4.8	2.28	90	27	30	7.8	ACIDOSIS	RIGHTMIDDLE AND LEFT LOWER ZONE PATCH	NORMAL	FUNGUS C/SET: ASPERGILLUS FLAVUS	NEG
55	910995	-	-	HMF ALTERED	NAD	NAD	NAD	11.5	4.3	88/8/4	160	34	NEG	1.45	15	0.84	136	3	0.37	4	1.99	188	91	93	7.2	ACIDOSIS	LEFT LZ PATCH	NORMAL	SPUTUM:KLEBSIELLA	NA
56	908077	-	-	HMF ALTERED	NAD	NAD	NAD	9.6	1.4	72/18/5/5	2.3	35	NEG	1.21	22	0.37	136	4.39	1.92	7.3	3.72	209	154	173	8.4	NORMAL	NORMAL	NORMAL	1	NEG
57	909520	-	-	HMFALTERED,INTUBATED	NAD	NAD	NAD	11	12.2	90/8/1	167	46	NA	1.23	20	0.3	139	4	0.34	6.2	3.4	362	49	70	7.2	NORMAL	NORMAL	NORMAL	1	NA
58	906529	Y	-	HMF ALTERED	NAD	NAD	NAD	7.8	16	93/5/2	215	63	NEG	1.17	92	0.8	132	4.8	0.37	7.5	3.6	63	24	29	8	NORMAL	NORMAL	ABNORMAL	1	NEG
59	907966	-	-	B/L PLANTAR EXTENSOR,NECK RIGIDITY,INTUBATED	NAD	NAD	NAD	8.8	11.5	83/9/7/1	150	52	NEG	1.23	17	0.31	121	4	0.8	6.5	3.6	70	51	70	8.2	RESPIRATORY ALKALOSIS	NORMAL	ABNORMAL	1	NEG
60	912943	-	-	HMF ALTERED	NAD	NAD	NAD	8.8	12.4	69/18/8/6	303	26	NEG	1.67	52	1.08	137	4	0.5	7	3.6	8	32	54	8.6	METABOLIC ACIDOSIS	NORMAL	NORMAL	1	NEG
61	906464	-	-	HMF ALTERED	NAD	NAD	NAD	7.9	9.8	68/18/10	549	38	NEG	1.78	27	2.31	128	3.28	0.49	5.9	2.79	116	23	16	8	NORMAL	NORMAL	NORMAL	WOUND SWAB:KLEBSIELLA	NEG
62	916640	-	-	HMF ALTERED	NAD	NAD	NAD	10.6	19.4	92/4/4	348	32	NEG	1.04	19	0.7	129	5	0.45	6.5	3.26	153	41	33	7.1	ACIDOSIS	NORMAL	NORMAL	URINE C/S: STAPH AUREUS	NEG
63	916165	-	-	HMF ALTERED	NAD	NAD	NAD	9.9	15.1	92/6/2	167	12	NEG	1.32	43	0.6	135	4	0.23	6	3.8	180	35	45	8	NORMAL	NORMAL	NORMAL	URINE C/S;E.COLI	NEG
64	606618	-	-	HMF ALTERED	NAD	B/LDIFFUSE CREPTS	NAD	8	19.1	89/9/1	238	110	NEG	1.78	60	2.93	141	3,5	0.38	6.2	2.96	417	40	25	5.9	ACIDOSIS	B/L PLEURAL EFFUUSION	NORMAL	URINE C/S:KLEBSIELLA	NA
65	885296	-	-	HMF ALTERED,POWER B/L LOWER LIMBS REDUCED	NAD	NAD	NAD	8.9	14.7	77/13/9/6	325	27	NEG	1.31	32	0.85	134	4.5	0.74	6.7	3.37	97	208	150	8	NORMAL	NORMAL	NORMAL	1	NEG
66	912359	Y	-	HMF ALTERED,NUCHAL RIGIDITY	NAD	NAD	NAD	11.1	16.2	84/6/10	442	34	NEG	1.2	98	0.86	134	4.89	0.29	7.7	3.49	54	105	32	7.9	NORMAL	RIGHT UPPER ZONE HAZINESS	ABNORMAL	1	NEG
67	919231	-	-	HMF ALTERED, NO MENINGEALSIGNS, B/LPLANTAR WITHDRAWL	NAD	B/L CREPTS HEARD	EPIGASTRIC MASS	10.1	16.5	84/6/7/3	178	45	NEG	1.21	137	5.71	137	5.4	1.31	7.2	2.21	269	30	11	8.9	NORMAL	NORMAL	NORMAL	1	NEG
68	919663	-	-	HMF ALTERED	NAD	NAD	NAD	9.8	8.7	65/27/10	267	12	NEG	1.32	22	0.55	147	4.1	0.2	6.3	3.19	48	19	13	9	ALKALOSIS	NORMAL	NORMAL	1	NEG
69	918746	-	-	DROWSY,MOVING LEFT HALF OF BODY MORE	NAD	B/L CREPTS HEARD	NAD	12	21.1	92/5/2.6	150	82	NEG	1.91	46	1.2	140	3.62	0.8	6.3	3.6	78	387	134	8.4	LACTICACIDOSIS	BLPLEURALEFFUSION AND ASPIRATION PNEUMONITIS	ABNORMAL	1	NEG
70	909443	-	-	HMF ALTERED,MENINGEAL SINGS PRESENT	NAD	NAD	NAD	11.5	9.2	97/2/1.2	318	37	NEG	1.2	31	0.57	137	4.3	0.4	5	2.62	126	52	91	8.6	NORMAL	ABNORMAL	NORMAL	1	NEG
71	919527	-	-	HMF ALTERED	NAD	RIGHT LZ CREPTS	NAD	8.4	13	80/18/2	353	90	NEG	1.1	62	0.8	142	4.1	0.79	5.5	2	74	26	16	9	NORMAL	RIGHT LOWER ZONE PATCH	NORMAL	SPUTUM C/S:KLEBSIELLA	NEG
72	920169	-	-	HMF ALTERED, B/LPLANTAR EXTENSOR	NAD	NAD	NAD	10.8	11.9	82/10/8	187	16	NEG	1.06	23	0.4	135	3.9	0.54	7	3.9	116	23	35	8	NORMAL	NORMAL	NORMAL	1	NEG
73	916538	-	-	DISORIENTED,LEFT PLANTAR EXTENSOR	NAD	NAD	NAD	10.6	13.7	78/22/10	222	45	NEG	1.32	33	0.97	136	4.2	0.2	6.9	3.62	226	70	55	6.8	NORMAL	NORMAL	NORMAL	1	NEG
74	394431	-	-	DROWSY,DISORIENTED,MOVING ALL 4 LIMBS	NAD	NAD	NAD	12.2	16.3	83/10/87	159	67	NEG	1.23	30	0.8	141	3.5	0.7	8	4.2	110	24	25	9.3	NORMAL	NORMAL	NORMAL	1	NEG
75	887278	-	-	MENINGEAL SIGNS,INTUBATED	NAD	NAD	NAD	7	16.9	89/12/5/2	118	68	NEG	1.42	30	1.6	139	4.1	5.6	3.2	80	64	78	51	7.2	NORMAL	NORMAL	NORMAL	1	NEG
76	919951	Y	-	MENINGEAL SIGNS PRESENT,HMF ALTERED	NAD	NAD	NAD	9.9	23.5	87/6/7/1	320	84	NEG	1.41	32	0.6	149	4.09	0.5	7.1	3.6	98	192	22	8.1	RESPIRATORY ALKALOSIS	NORMAL	NORMAL	1	NA
77	919674	-	-	HMF NORMAL	NAD	NAD	NAD	10.8	24.5	70/20/10	97	17	NEG	1.09	43	1.28	141	3.8	0.53	6	2.63	137	30	31	8.9	NORMAL	NORMAL	NORMAL	URINE C/S: E.COLI	NEG
78	920784	-	-	MENINGIAL SINGS,DROWSY,NOT FOLLOWING COMMANDS	NAD	NAD	NAD	11.2	12.6	64/22/11/2	338	65	NEG	1.15	21	0.69	130	3.96	0.55	6.4	3.84	63	25	32	7.8	NORMAL	NORMAL	NORMAL	1	NEG
79	774837	-	-	HMF ALTERED,B/LPLANTAR MUTE	ABNORMAL	B/LCREPTS PRESENT	NAD	7.8	42	95/3/2	167	89	NEG	1.01	72	3.26	129	4.6	0.54	5.6	2.82	121	67	89	7.2	ACIDOSIS,TYPE 1 RF	RIGHT LZ HAZINESS	ABNORMAL	URINE/SPUTUM C/S: KLEBSIELLA	NEG
80	933611	-	-	HMF ALTERED	ABNORMAL	NAD	NAD	11.6	14	90/3/6	438	120	NEG	1.39	176	2.07	139	8	0.68	7.2	2.56	161	99	62	9	ACIDOSIS,HYPERKALEMIA	NORMAL	ABNORMAL	WOUND SWAB :MRSA	NA
81	920626	-	-	DROWSY,B/LPLANTAR UPGOING	NAD	NAD	NAD	13	17	88/6/7	296	9	NEG	1.26	32	0.8	134	3.8	0.48	7.3	4.3	80	35	77	9.2	NORMAL	ABNORMAL	ABNORMAL	1	NEG
82	760453	-	-	HMF NORMAL	NAD	NAD	NAD	7.7	13	73/17/10	184	24	NEG	1.09	74	3.26	131	5.3	0.58	7.3	3.25	789	67	20	9	NORMAL	NORMAL	NORMAL	1	NEG
83	922167	-	-	HMF ALTERED	NAD	NAD	NAD	11	15	89/9/1	170	88	NEG	1.27	24	0.6	138	3.9	0.5	6.6	3.7	80	33	67	8	NORMAL	NORMAL	NORMAL	1	NEG
84	923527	-	-	HMF ALTERED	ABNORMAL	NAD	NAD	10.1	31.5	89/6/7	373	13	NEG	1.11	50	1.22	144	4.41	0.51	6.6	3.33	83	26	16	8.7	ACIDOSIS	B/L DIFFUSE INFILTRATES	ABNORMAL	1	NA
85	923849	-	-	HMF ALTERED	NAD	NAD	NAD	12.3	23.2	76/13/10	150	54	NEG	0.9	67	1.71	142	4.3	0.34	7.3	4.1	90	45	33	7	ACIDOSIS	NORMAL	NORMAL	1	NA

S.NO	MRD NO	Leptospira	Dengue	Scrub Typhus	Other Investigations	CSF CYTOLOGY_TLC	NEUTROPHILS	LYMPHOCYTES	GLUCOSE	S. GLUCOSE	PROTEIN	CHLORIDE	GRAM STAIN	INDIA INK PREPARATION	BACTERIAL ANTIGEN	CULTURE SENSITIVITY	ADA	CBNAAT	HSV PCR	CT HEAD_FOCAL HYPODENSITIES	CT HEAD HYDROCEPHALUS	CT HEAD_DIFFUSE CEREBRAL OEDEMA	CT HEAD INFARCT	CT HEAD OTHERS	CT HEAD NORMAL	MRI LEPTOMENINGITIS	MRI VASCULITIC INFARCT
44	906511	NEG	NEG	NEG	PCT 88,2D ECHO: EF 22%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
45	905849	NA	NA	NA	-	120	10	90	88	142	64	112	-	-	-	-	4.2	-	NEGATIVE	N	N	N	N	N	N	N	N
46	907879	NA	NA	NA	URINE R/E:80 PUS CELLS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
47	869413	NA	NA	NA	-	25	5	95	16	87	88	107	NO GROWTH	-	-	-	11	-	-	N	Y	N	N	N	N	Y	N
48	903037	NA	NA	NA	-	20	10	90	89	92	57	139	-	-	-	-	0.58	-	POSITIVE	N	N	N	N	N	N	N	N
49	909322	NA	NA	NA	-	2	0	0	115	167	92	110	-	-	-	-	1.34	-	POSITIVE	Y	N	N	N	N	N	N	N
50	899137	NA	NA	NA	GHK EF 36%	180	30	70	79	140	109	110	-	-	-	-	12.2	POSITIVE	-	N	N	N	N	N	N	Y	Y
51	904335	NEG	NEG	POSITIVE	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
52	792833	NA	NA	NA	L3,L4 MARROW OEDEMA	65	5	95	34	98	121	108	-	-	-	-	16	-	-	N	N	N	N	N	Y	Y	Y
53	908748	NA	NA	NA	-	160	10	90	45	99	143	115	-	-	-	-	17.2	POSITIVE	-	N	N	N	N	N	N	Y	N
54	910213	NA	NA	NA	PCT 2.28	2	0	0	117	178	45	108	-	-	-	-	1.08	-	-	N	N	N	N	N	Y	N	N
55	910995	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
56	908077	NA	NA	NA	-	20	10	90	46	125	164	-	-	-	-	-	29.2	POSITIVE	-	N	N	N	N	N	N	N	y
57	909520	NA	NA	NA	-	5	0	100	39	187	1829	102	-	-	-	-	161	-	NEGATIVE	Y	N	N	N	N	N	N	N
58	906529	NA	NA	NA	-	80	10	90	43	110	205	108	-	-	-	-	26	-	-	N	N	N	N	N	N	Y	Y
59	907966	NA	NA	NA	-	90	10	90	32	89	167	108	-	-	-	-	-	POSITIVE	-	N	N	N	Y	N	N	Y	Y
60	912943	NA	NA	NA	-	1500	95	5	22	90	88	113	-	-	-	-	6	-	-	N	N	N	N	N	N	Y	N
61	906464	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
62	916640	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
63	916165	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
64	606618	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
65	885296	NA	NA	NA	-	45	0	100	47	114	112	116	-	-	-	-	17	-	-	N	N	N	N	N	N	Y	Y
66	912359	NEG	NEG	NEG	-	70	10	90	54	89	87	110	-	-	-	-	21	-	-	Y	N	N	N	N	N	Y	Y
67	919231	NEG	NEG	NEG	USG: ACUTE PANCREATITIS/LIPASE: 1400	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
68	919663	NA	NA	NA	-	15	10	90	63	204	48	122	-	-	-	-	-	-	NEGATIVE	N	N	N	N	N	N	N	N
69	918746	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	DIFFUSE CEREBRAL ATROPHY	N	N	N
70	909443	NA	NA	NA	-	250	20	80	25	111	13	97	-	-	-	-	22	-	-	N	Y	N	N	N	N	Y	Y
71	919527	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
72	920169	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
73	916538	NA	NA	NA	HEP B RECATIVE	80	10	90	76	108	75	125	-	-	-	-	1.96	-	NEGATIVE	N	N	N	N	NA	N	Y	N
74	394431	NA	NA	NA	-	20	10	90	65	140	52	121	-	-	-	-	0.4	-	POSITIVE	N	N	N	N	N	Y	N	N
75	887278	NA	NA	NA	-	30	10	90	40	110	96	102	-	-	-	-	-	POSITIVE	-	Y	N	N	N	N	N	Y	Y
76	919951	NA	NA	NA	-	20	0	100	27	110	60.6	113	-	-	-	-	0.78	-	-	N	Y	N	N	N	N	Y	Y
77	919674	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
78	920784	NA	NA	NA	-	40	10	90	19	168	137	111	-	-	-	-	29	POSITIVE	-	N	N	N	N	N	Y	Y	Y
79	774837	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
80	933611	NA	NA	NA	NCCT KUB :LEFT PYELITIS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
81	920626	NA	NA	NA	-	30	60	40	44	98	133	110	-	-	-	-	-	-	POSITIVE	N	N	N	N	N	N	N	N
82	760453	NA	NA	NA	FUNGITELL POSITIVE	5	0	0	82	110	56	108	-	-	-	-	3	-	-	N	N	N	N	N	N	N	N
83	922167	NA	NA	NA	-	80	10	90	77	89	234	109	-	-	-	-	35.9	POSITIVE	-	Y	N	N	N	N	N	Y	N
84	923527	NA	NA	NA	-	2	0	0	201	227	38	108	-	-	-	-	2.1	-	-	N	N	N	N	N	Y	N	N
85	923849	NA	NA	NA	URINE R/E 60PUS CELLS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N

S.NO	MRD NO	MRI_RING ENHANCING LESIONS	MRI_MENINGIOENCEPHALITIS/ ENCEPHALITIS	MRI HYDROCEPHALUS	MRI_DIFFUSE CERBRAL OEDEMA	MRI NORMAL	MRI_OTHERS	EEG (if needed)	TUBERCULOUS MENINGITIS	2.VIRAL	3.DENGUE ENCEPHALOPATHY/ ENCEPHALITIS	4.PYOGENIC	5.MALARIA	6.SCRUB RELATED ENCEPHALOPATHY	7.SEPTIC ENCEPHALOPATHY	POST COVID ENCEPHALOPATHY	LEPTOSPIRA INDUCED ENCEPHALOPATHY	OTHERS	TREATMENT GIVEN	OUTCOME 1. RECOVERED, 2. DAMA,
44	906511	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE/VENTILATOR	2
45	905849	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRALS/ ANTI EPILEPTIC/ATEROIDS	1
46	907879	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
47	869413	Y	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROID/ANTI EPI	1
48	903037	N	Y	N	N	N	FEATURES S/O HERPES ENCEPHALITIS	DIFFUSE ENCEPHALOPATHY	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRALS/ANTIEPILEPTICS/STEROIDS	1
49	909322	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRAL/IV ANTIBIOTICS,ANTIEPILEPTICS,MECHANICAL VENTILATORY SUPPORT	3
50	899137	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDSTOLVAPTAN	2
51	904335	N	N	N	N	N	B/LPOSTERIOR CIRCULATION MULTIFOCAL INFARCTS	-	N	N	N	N	N	Y	N	N	N	N	ATT/DOXYCYCLINE/STEROIDS	1
52	792833	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROID	1
53	908748	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROID	1
54	910213	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/VENTILATOR/INOTROPES	2
55	910995	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	3
56	908077	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	MODIFIED ATT	1
57	909520	N	N	N	N	N	CYTOTOXICOEDEMA B/L TEMPORAL LOBES	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
58	906529	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT,STEROID/BACTRIM DS	1
59	907966	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	3
60	912943	N	N	N	N	N	-	-	N	N	N	STREPTOCOCCUS PNEUMONIAE	N	N	N	N	N	N	IV ANTIBIOTICS/STEROIDS	2
61	906464	N	N	N	N	N	CYTOTOXIC OEDEMAIN RIGHT CORONA RAIATA	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBOTICS/DEBRIDEMENT OF RIGHT DIABETIC FOOT	1
62	916640	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	2
63	916165	N	N	N	N	N	-	MILD DIFFUSE ENCEPHALOPATHY	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS	1
64	606618	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS,ANTIEPILEPTIC	3
65	885296	N	N	N	N	N	E/O POTT'S SPINE	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	2
66	912359	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROID/ANTI OEDEMA	1
67	919231	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV FLUIDS/INOTROPES/SLED	3
68	919663	N	Y	N	N	N	? ATYPICALPRES	-	N	Y	N	N	N	N	N	N	N	N	IB ANTIVIRALS/IV ANTIBIOTICS/ANTI EPILEPTIC	1
69	918746	N	N	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIBIOTICS,ACYCLOVIR,ANTIEPILLEPTICS	3
70	909443	Y	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/IV ANTIBIOTICS	2
71	919527	N	N	N	N	N	-	-	N	N	N	N	N	N	N	N	N	N	IV ANTIBITOCS/SUPPORTVE	1
72	920169	N	N	N	N	N	PRES	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ANTIEPILEPTICS	1
73	916538	N	N	N	N	N	NA	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIBIOTICS,ACYCLOVIR,ANTIEPILLEPTICS	1
74	394431	N	N	N	N	Y	-	-	N	Y	N	N	N	N	N	N	N	N	ACYCLOVIR/ANTIEPLEPTICS	1
75	887278	N	N	N	N	N	SUBDURAL COLLECTIONS	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/ATIBIOTICS	1
76	919951	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	2
77	919674	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
78	920784	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
79	774837	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/VENTILATOR	3
80	933611	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	2
81	920626	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	STEROID PULSE,ANTIEPILEPTIC,ACYCLOVIR	3
82	760453	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBITOICS/STEROIDS	1
83	922167	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
84	923527	N	N	N	N	Y	-	MODERATE DIFFUSE ENCEPHALOPATHY	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS/SUPPORTIVE	2
85	923849	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/VENTILATOR/SUPPORTIVE	2

S.NO	MRD NO	DOA	AGE	SEX	RELIGION	OCCUPATION	CHIEF COMPLAINTS FEVER	ALTERED SENSORIUM	HEADACHE	VERTIGO	BLURRING VISION	ABNORMAL BODY MOVEMENT	WEAKNESS IN ANY PART OF	VOMITING	OTHERS	PAST MEDICAL ILLNESS HISTORY			FAMILY HISTORY	PERSONAL HISTORY				SBP	DBP	TEMP	PULSE RATE	RR	PALOR	GCS	CLUBBING	ICTERUS	EDEMA	PETECHIAE / PURPURA
																TYPE 2 DM	HYPERTENSION	OTHERS		ALCOHOL	SMOKING	SUBSTANCE ABUSE	NONE										
86	923746	27/4/20	40	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	-	-	HIV/MILIARY TB	NIL	-	-	-	Y	90	60	100	98	18	Y	12	-	-	-	-
87	922325	28/4/20	37	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	100	60	99.8	108	16	-	11	-	-	-	-
88	924790	2-5-20	67	M	SIKH	BUSINESS	Y	Y	N	N	N	N	N	N	SHORTNESS OF BREATH	Y	Y	-	NIL	Y	-	-	-	80	60	99	90	18	-	12	-	-	-	-
89	923505	4-5-20	27	M	SIKH	BUSINESS	Y	Y	Y	N	N	N	N	Y	MALARIA 1 M BACK	-	-	-	NIL	-	-	-	Y	100	60	102	100	21	-	10	-	-	-	-
90	923819	4-5-20	38	M	MUSLIM	LABOUR	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	99	98	20	-	12	-	-	-	-
91	925545	11-5-20	58	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	PAIN ABDOMEN	-	-	-	NIL	Y	-	-	-	70	40	102	156	22	-	9	-	-	-	-
92	926731	16/5/20	24	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	100	60	98	89	18	-	8	-	-	-	-
93	928138	21/5/20	73	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	CA PROSTATE	NIL	-	-	-	Y	80	60	101	110	22	-	10	-	-	-	-
94	527028	21/5/20	63	M	HINDU	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	-	-	-	PARKINSON'S	NIL	-	-	-	Y	90	60	99	112	26	-	11	-	-	-	-
95	927159	26/5/20	55	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	Y	-	NIL	-	-	-	Y	120	70	101	98	17	-	9	-	-	-	-
96	920342	8-6-21	63	M	SIKH	SERVICE	Y	Y	N	N	N	Y	N	N	-	Y	-	CAD/OLD KOCH;S	NIL	-	-	-	Y	110	60	99	109	18	-	9	-	-	-	-
97	934046	11-6-21	40	F	HINDU	DOCTOR	Y	Y	N	N	N	N	N	N	PAIN ABDOMEN,DECREASED URINE OUTPUT	Y	-	-	NIL	-	-	-	Y	80	40	97.2	136	22	-	11	-	-	-	-
98	934446	13/6/20	68	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	DECREASED URINE OUTPUT	Y	-	-	NIL	-	-	-	Y	90	50	100	98	18	-	9	-	-	-	-
99	903486	13/6/20	80	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	DECREASED URINE OUTPUT	-	-	POST RSA/BEDRIDDEN	NIL	Y	-	-	-	100	70	101	116	22	Y	10	-	-	-	-
100	931917	15/6/20	22	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	-	-	-	TB IN YOUNGER SISTER	-	-	-	Y	100	60	99	72	16	-	3	-	-	-	-
101	929572	18/6/20	18	F	HINDU	STUDENT	Y	Y	N	N	N	N	N	N	MULTIPLE SWELLINGS OVER BODY	-	-	-	NIL	-	-	-	Y	90	70	98.2	88	18	-	12	-	-	-	-
102	935412	19/6/20	18	F	HINDU	STUDENT	Y	Y	N	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	100	60	101	118	22	-	9	-	-	-	-
103	936136	20/6/20	88	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	Y	-	-	-	POST RSA/BEDRIDDEN	NIL	-	-	-	Y	100	70	101	112	20	-	12	-	-	-	-
104	934749	20/6/20	30	F	HINDU	DEPENDENT	Y	Y	Y	N	Y	N	N	Y	-	-	-	-	NIL	-	-	-	Y	110	80	99.6	80	17	Y	12	-	-	-	-
105	933086	21/6/20	62	F	SIKH	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	80	40	98.5	123	15	Y	8	Y	-	-	-
106	935006	21/6/20	22	M	SIKH	STUDENT	Y	Y	N	N	N	N	N	N	LOOSE STOOLS	-	-	-	NIL	-	-	-	Y	60	40	100	99	14	-	12	-	-	-	-
107	937358	24/6/20	55	F	SIKH	DEPENDENT	Y	Y	N	Y	N	N	N	N	SHORTNESS OF BREATH	Y	-	-	NIL	-	-	-	Y	110	70	100	118	18	Y	12	-	-	-	-
108	936387	24/6/20	49	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	-	-	RA	NIL	-	-	-	Y	110	70	99	108	20	-	12	-	-	-	-
109	938278	27/6/20	45	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	Y	DREASED URINE OUTPUT	Y	-	-	NIL	-	-	-	Y	100	60	100	110	20	-	11	-	-	-	-
110	630907	30/6/20	43	M	SIKH	BUSINESS	Y	Y	N	N	N	N	N	N	BACKPAIN	-	Y	OLD KOCH'S	NIL	-	-	-	Y	130	90	99.4	110	22	-	12	-	-	-	-
111	939085	3-7-20	46	M	HINDU	BUSINESS	Y	Y	N	N	N	Y	N	Y	-	-	-	-	NIL	-	-	-	Y	110	70	100	108	21	Y	8	-	-	-	-
112	942598	4-7-20	18	F	SIKH	STUDENT	Y	Y	Y	N	N	N	N	N	-	-	-	-	H/O PULMONARY TB IN YOUNGER BROTHER	-	-	-	Y	110	70	101	112	21	Y	12	-	-	-	-
113	940763	6-7-20	73	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	-	Y	Y	-	NIL	-	-	-	Y	100	60	99.9	104	8	-	12	-	-	-	-
114	673574	8-7-20	61	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	Y	-	NIL	-	Y	-	-	90	50	99.8	108	18	Y	11	-	-	-	-
115	940295	13/7/20	82	Y	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	SEIZURE DISORDER	NIL	-	-	-	Y	50	40	101	126	26	-	5	-	-	-	-
116	942461	13/7/20	45	M	HINDU	BUSINESS	Y	Y	Y	N	Y	N	N	Y	-	-	-	-	NIL	-	-	-	Y	120	70	99	112	18	-	10	-	-	-	-
117	934668	16/7/20	59	M	HINDU	BUSINESS	Y	Y	Y	N	N	Y	N	Y	SHORTNESSOF BREATH	Y	-	-	PULMONARY TB IN SON	-	-	-	Y	100	70	100	113	27	-	12	-	-	-	-
118	945392	20/7/20	77	M	HINDU	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	-	-	HYPOTHYROID/ PARKINSON'S	NIL	-	-	-	Y	70	40	101	167	28	Y	6	-	-	-	-
119	698844	22/7/20	80	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	Y	DECREASED URINE OUPUT	Y	-	CAD	NIL	-	-	-	Y	70	40	99	108	22	-	10	-	-	-	-
120	75711	23/7/20	80	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	98	100	20	Y	12	-	-	-	-
121	942598	25/7/20	18	F	SIKH	STUDENT	Y	Y	Y	N	Y	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	99	64	18	-	11	-	-	-	-
122	942631	28/7/20	45	M	HINDU	SERVICE	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	98.9	88	22	-	10	-	-	-	-
123	949182	31/7/20	27	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	HEMATEMESIS	-	-	-	NIL	-	-	-	Y	90	40	100	122	26	-	9	-	-	-	-
124	949989	1-8-20	26	M	SIKH	STUDENT	Y	Y	N	N	N	N	Y	N	-	-	-	-	NIL	-	-	-	Y	100	60	101	115	22	-	6	-	-	-	-
125	947989	4-8-20	78	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	LOOSE STOOLS, DECREASED URINE OUTPUT	Y	Y	-	NIL	-	-	-	Y	70	40	99	137	28	Y	7	-	-	-	-
126	946169	5-8-20	54	F	HINDU	DEPENDENT	Y	Y	Y	N	N	Y	Y	N	-	-	-	OLD KOCH'S	NIL	-	-	-	Y	120	70	97.8	76	16	-	12	-	-	-	-
127	951754	6-8-20	48	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	COUGH/SHORTNESS OF BREATH	-	-	OLD KOCH'S	NIL	-	-	-	Y	110	70	99.8	108	32	-	11	-	-	-	-

S.NO	MRD NO	LYMPHADENOPATHY	CYANOSIS	CENTRAL NERVOUS SYSTEM	CVS	RS	P/A	HB	TLC	DLC	PLT	ESR	PBF MALARIA	PTI-INR	UREA	CREATININE	NA	K	S. BILIRUBIN	TOTAL PROTEIN	ALBUMIN	ALP	SGOT	SGPT	CALCIUM	ABG	CXR	ECG	BLOOD C/S/URINE C/S	Malaria Antigen
86	923746	Y	-	HMF ALTERED	NAD	NAD	ABDOMEN DISTENDED	6.8	5.4	84/8/6	332	45	NA	1.33	26	0.6	144	3.6	52	7.4	3.04	98	27	23	8	NORMAL	NORMAL	NORMAL	1	NA
87	922325	-	-	NECK RIGIDITY,ALTERED HMF	NAD	NAD	NAD	14	9.5	65/25/2	165	45	NEG	1.34	34	0.5	133	3.2	0.8	6	3.02	110	54	74	9	NORMAL	NORMAL	NORMAL	1	NEG
88	924790	-	-	HMF ALTERED	NAD	B/L AE REDUCED	NAD	8	9.3	74/14/10	130	20	NEG	1.19	108	5.79	135	4	0.6	5.5	2.49	104	40	30	7.8	ACIDOSIS	B/LLZ HAZINESS	ABNORMAL	URINE C/S:ENTEROCOCCUS FAECIUM	NEG
89	923505	-	-	DROWSY,NO VERBAL RESPONSE,B/L PLANTAR EXTENSOR	NAD	NAD	NAD	9.2	7.4	84/12/5201	201	10	NEG	1.13	43	0.54	142	3.9	0.46	7	4	137	66	51	8	NORMAL	MILIARY MOTTLING	NORMAL	1	NEG
90	923819	Y	-	DROWSY,POWER LESSIN LEFT UPPER LIMB	NAD	NAD	NAD	8.9	6.2	82/7/0.5	239	80	NEG	1.23	36	0.33	135	3.4	0.8	7.2	4.6	83	22	63	8.2	NORMAL	NORMAL	NORMAL	1	NEG
91	925545	-	-	HMF ALTERED	NAD	NAD	PALPABLE LIVER,IRREGULAR BORDER	8.9	23	88/10/2	190	34	NEG	1.89	192	3.3	133	4	5.8	6.2	3.37	380	44	72	8	ACIDOSIS	NORMAL	NORMAL	1	NEG
92	926731	-	-	HMF ALTERED	NAD	NAD	NAD	11.6	13.3	82/16/8	341	14	NA	1.33	24	0.2	134	3	0.78	6.9	3.6	88	45	90	9.1	NORMAL	NORMAL	NORMAL	1	NEG
93	928138	-	-	HMF ALTERED	NAD	B/L SPASM	NAD	6.9	6.4	78/22	100	100	NEG	0.89	39	1.02	144	4	1.05	5	2.89	1408	45	8	9.2	ALKALOSIS	NORMAL	NORMAL	URINE C/S: E.COLI	NEG
94	527028	-	-	HMF ALTERED	NAD	NAD	NAD	10.9	11.5	75/20/5	248	12	NA	1.36	18	0.62	134	3.9	0.95	6.1	3.11	81	38	42	8.2	ACIDOSIS	NORMAL	NORMAL	URINE C/S:PSEUDOMONAS	NA
95	927159	-	-	MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	7.8	15.7	84/11/6	29	2	NEG	1.27	82	1.98	130	5.2	0.52	4.2	2.58	32	38	20	9	ABNORMAL	ABNORMAL	NORMAL	URINE C/S: CANDIDA	NEG
96	920342	Y	-	HMF ALTERED, MOVING ALL 4 LIMBS, INTUBATED	NAD	NAD	NAD	10.6	24	87/8/3	100	23	NEG	1.43	82	2.14	132	4.9	1.39	5.6	2.11	364	225	112	8.1	NORMAL	NORMAL	NORMAL	1	NEG
97	934046	-	-	HMF ALTERED	NAD	NAD	DISTENDED,GUARDING,RIGIDITY	9.7	42	86/10/4	28	67	NEG	1.19	104	4.84	142	5	1	4.6	2.18	149	26	8	8.9	ACIDOSIS	NORMAL	NORMAL	VAG SWAB: E.COLI	NEG
98	934446	-	-	HMF ALTERED	NAD	RIGHT LZ CREPTS	NAD	8.5	48.5	75/8/12	251	24	NEG	1.1	116	5.46	137	3.8	0.25	5.3	2.26	176	20	7	8.1	ACIDOSIS	RIGHT LZ HAZINESS	NORMAL	URINE C/S:CANDIDA	NEG
99	903486	-	-	HMF ALTERED, B/LPLANTAR FLEXOR	NAD	B/L LZ AE REDUCED	NAD	8.4	15.8	94/3/2/1	641	34	NEG	1.21	92	1.52	150	4	0.42	7	2.99	129	28	32	8.4	ACIDOSIS	B/L PLEURAL EFFUSION	NORMAL	URINE C/S: E.COLI	NEG
100	931917	-	-	HMF ALTERED, B/L PLNTAR MUTE, B/L PUPILS FIXED AND DIATED	NAD	B/L DECREASED AIR ENTRY	HEPATOMEGALY	8.3	10.4	68/24/2	245	98	NA	1.4	21	0.19	138	3.7	0.38	6.3	3.21	100	55	32	9.2	METABOLIC ACIDOSIS	RIGHT PLEURAL EFFUSION	NORMAL	1	NEG
101	929572	-	-	HMF ALTERED	NAD	NAD	NAD	9.6	5.99.3	82/18/8/2	183	47	NA	1.37	30	0.6	132	4.2	0.2	6.3	3.2	70	12	24	8	NORMAL	MILIARY SHADOWS	NORMAL	1	NEG
102	935412	-	-	HMF ALTERED,MENINGEAL SIGNS	NAD	NAD	NAD	8.9	12	68/22/8	179	99	NA	1.34	20	0.4	135	4	0.5	6	3.65	88	21	56	8	NORMAL	NORMAL	NORMAL	1	NA
103	936136	-	-	HMF ALTERED	ABNORMAL	RIGHT LZ CREPTS	NAD	12.2	10.2	80/12/8	156	27	NEG	1.09	14	0.6	134	5	0.45	6.8	3.4	67	37	38	8.2	NORMAL	RIGHT LZ PATCH	ABNORMAL	1	NEG
104	934749	-	-	HMF ALTERED	NAD	NAD	NAD	12.7	11.7	68/22/5/2	156	33	NEG	1.13	50	0.6	129	4	0.6	6.4	3.2	88	81	75	7.5	NORMAL	NORMAL	NORMAL	1	NA
105	933086	-	-	NECK RIGITY,INTUBATED	NAD	NAD	NAD	9.6	14.2	75/22/3.6	242	33	NEG	1.11	30	0.5	133	4	0.48	7.9	4	107	163	84	7.8	ABNORMAL	ABNORMAL	ABNORMAL	1	NEG
106	935006	-	-	HMF ALTERED,NECK RIGIDITY	NAD	NAD	NAD	11.5	6.9	52/36/9.7/0.	249	82	NA	1.32	20	0.83	140	3.6	0.7	6.2	3.6	80	52	71	8.1	NORMAL	NORMAL	NORMAL	1	NEG
107	937358	-	-	HMF ALTERED	NAD	RIGHT BRONCHIAL BREATHING/CREPTS	NAD	7.9	6.9	69/28/3	181	32	NA	1.1	23	0.69	123	4	0.2	7	4.2	66	23	54	9.2	TYPE 1 RF	LEFT LOBAR PNEUMONITIS	NORMAL	SPUTUM C/S: KLEBSIELLA	NA
108	936387	-	-	HMF ALTERED, B/L PLANTAR WITHDRAWL,NECK RIGIDITY	NAD	NAD	NAD	9.5	5.6	72/16/8	145	88	NA	1.45	24	0.8	120	4	0.03	7.2	4.12	88	24	27	9	NORMAL	NORMAL	NORMAL	1	NA
109	938278	-	-	HMF ALTEERED,MOVING ALL 4 LIMBS	NAD	NAD	NAD	12.5	17.3	70/21/3	205	81	NA	1.53	48	2.38	132	4.5	1,86	6.8	3.53	295	55	53	7.6	ACIDOSIS	B/L DIFFUSE INFILTRATES	NORMAL	1	NEG
110	630907	Y	-	HMF ALTERED	NAD	NAD	NAD	12	5.8	61/28/9	207	6	NEG	1.06	19	0.81	134	4.8	0.9	6.8	3.2	88	21	31	8.2	NORMAL	NORMAL	NORMAL	1	NEG
111	939085	-	-	HMF ALTERED,INTUBATED,B/L PLANTAR MUTE	NAD	NAD	NAD	7.4	8.2	65/22/8	148	87	NEG	1.23	30	0.63	148	2.6	0.57	7.4	3.71	79	114	46	8.3	NORMAL	NORMAL	NORMAL	1	NEG
112	942598	-	-	HMF ALTERED	NAD	NAD	NAD	8.1	11.1	88/18/2/3	180	82	NEG	1.3	24	0.3	133	4.3	0.2	6.8	3.46	71	12	24	8.1	NORMAL	NORMAL	NORMAL	1	NEG
113	940763	-	-	HMF ALTERED	ABNORMAL	NAD	NAD	10.7	14.2	78/20/10	235	43	NA	1.19	99	1.08	138	4	0.45	6.1	3.1	150	34	56	8.5	NORMAL	NORMAL	ABNORMAL	1	NA
114	673574	-	-	HMF ALTERED	NAD	NAD	NAD	7.3	10.6	81/18/1	362	87	NA	1.09	115	6.19	133	3.8	0.26	6.3	3.1	46	32	27	8	ACIDOSIS	NORMAL	NORMAL	URINE C/S: KLEBSIELLA	NEG
115	940295	-	-	HMF ALTERED,INTUBATED,B/L PLANTAR MUTE	NAD	NAD	NAD	15.9	8	83/8/8/1	253	15	NEG	1.3	52	1.15	132	4.5	1.69	6.3	2.75	137	34	38	8.06	NORMAL	NORMALLEFT MZ OPCITY	NORMAL	1	NEG
116	942461	-	-	HMF ALTERED,B/LPLANTAR EXTENSOR	NAD	NAD	NAD	12.3	12.4	73/12/5	167	77	NEG	1.34	23	0.5	133	4	0.06	5.4	2.76	89	34	45	6.9	RESPIRATORY ALKALOSIS	B/L LZ HAZINESS	ABNORMAL	1	NEG
117	934668	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	B/L CREPTS HEARD	NAD	10.2	2	90/8/2	189	77	NEG	1.23	48	4.1	138	4.9	0.93	6.1	2.89	189	67	87	9.2	NORMAL	B/L INFILTRATES	NORMAL	1	NEG
118	945392	-	-	HMF ALTERED, B/L PLANTARMUTE	NAD	NAD	NAD	8.3	14.2	75/15/10	391	54	NA	0.78	39	1.78	145	4.5	0.53	5.6	2.59	103	35	18	9.5	ACIDOSIS	RIGHT UZN PATCH	NORMAL	1	NA
119	698844	-	-	HMF ALTERED	NAD	NAD	NAD	10.6	19	82/16/2	160	23	NA	1.56	22	0.4	137	4	0.34	6.2	2.8	189	34	45	7.1	ACIDOSIS	NORMAL	NORMAL	URINE C/S:E.COLI	NEG
120	75711	-	-	HMF ALTERED	NAD	NAD	NAD	7.3	19.2	89/11/	122	110	NEG	1.34	48	4.92	136	5	0.67	6.5	3.1	199	23	78	8.2	ACIDOSIS	B/L DIFFUSE INFILTRATES	NORMAL	URINE C/S: PROTEUS	NEG
121	942598	-	-	HMF ALTERED, MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	7.8	11.2	80/12/8	189	90	NEG	0.98	34	0.23	140	3.6	0.43	5.4	2.45	98	65	34	9	NORMAL	MILIARY SHADOWS	NORMAL	1	NEG
122	942631	Y	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	12.1	9.1	93/4/4	110	82	NEG	1.33	36	0.36	144	3.8	0.8	7.2	3.02	88	118	298	8	METABOLIC ACIDOSOS	NORMAL	NORMAL	1	NEG
123	949182	-	-	RIGHT HEMIPARESIS,HMF ALTERED	NAD	NAD	NAD	7.8	300	33/10/2	29	102	NEG	1.23	120	1.4	131	3.6	0.7	5.9	3.2	80	102	118	9.1	METABOLIC/RESPIRATORY ACIDOSIS	NORMAL	NORMAL	1	NEG
124	949989	-	-	HMF ALTERED,RIGHT HEMIPARESIS	NAD	NAD	NAD	9.8	30.2	78/21/2	383	12	NEG	1.22	18	0.4	136	3.8	0.34	6.8	3.23	78	32	34	9	LACTIC ACIDOSIS	LEFTMIDDLEZONEPATCH	NORMAL	ET C/S:KLEBSIELLA	NEG
125	947989	-	-	HMF ALTERED	ABNORMAL	B/LDIFFUSE CONDUCTED SOUNDS	NAD	7.9	14.5	80/15/5	90	53	NA	1.18	115	5.91	144	5	0.5	4	2.13	140	35	44	8.3	ACIDOSIS	B/L DIFFUSE INFILTRATES	ABNORMAL	BLOOD C/S: BURKHOLDERIA	NEG
126	946169	-	-	CONFUSED,LEFT SIDE POWER DECREASED,LEFT PLANTAR EXTENSOR	NAD	NAD	NAD	14	10.3	85/8/6.4	269	28	NEG	1.09	36	0.51	137	3.8	0.8	7.2	4.1	90	141	249	7.6	NORMAL	ATELECTTIC BANDS	NORMAL	-	NEG
127	951754	-	-	HMF ALTERED	NAD	B/L CREPTS	NAD	9.1	12.8	72/12/6	345	89	NEG	0.98	52	1.06	139	4.7	0.15	5.6	2.99	168	20	17	9	TYPE 1 RF	ARDS	NORMAL	1	NEG

S.NO	MRD NO	Leptospira	Dengue	Scrub Typhus	Other Investigations	CSF CYTOLOGY_TLC	NEUTROPHILS	LYMPHOCYTES	GLUCOSE	S. GLUCOSE	PROTEIN	CHLORIDE	GRAM STAIN	INDIA INK PREPARATION	BACTERIAL ANTIGEN	CULTURE SENSITIVITY	ADA	CBNAAT	HSV PCR	CT HEAD_FOCAL HYPODENSITIES	CT HEAD HYDROCEPHALUS	CT HEAD_DIFFUSE CEREBRAL OEDEMA	CT HEAD INFARCT	CT HEAD OTHERS	CT HEAD NORMAL	MRI LEPTOMENINGITIS	MRI VASCULITIC INFARCT
86	923746	NA	NA	NA	CD4 33/HRCT CHEST: MILIARY MOTTLING	80	20	80	31	89	171	98	-	-	-	-	-	POSITIVE	-	N	N	N	N	N	N	NA	NA
87	922325	NA	NA	NA	-	60	10	90	48	110	53	134	-	-	-	-	9.11	-	-	N	N	N	N	N	N	Y	Y
88	924790	NA	NA	NA	PCT 3.01	2	0	0	114	156	34	118	-	-	-	-	1.25	-	-	N	N	N	N	N	N	N	N
89	923505	NA	NA	NA	-	20	10	90	38	183	100	111	-	-	-	-	16.1	POSITIVE	-	Y	N	N	N	N	N	Y	N
90	923819	NA	NA	NA	-	10	0	100	22	88	75	50	-	-	-	-	32	-	-	N	N	N	N	N	N	Y	N
91	925545	NA	NA	NA	USG - LIVER ABSCESSES	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
92	926731	NA	NA	NA	-	60	10	90	19	89	514	112	-	-	-	-	6.5	POSITIVE	-	N	Y	N	N	N	N	Y	N
93	928138	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
94	527028	NA	NA	NA	PCT 17	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
95	927159	NEG	NEG	NEG	-	85	30	70	34	90	86	100	-	-	-	-	3.7	-	-	N	N	N	N	N	N	N	N
96	920342	NA	NEG	NA	-	600	10	90	124	203	235	118	-	-	-	-	17	-	NEGATIVE	N	N	N	N	N	Y	Y	N
97	934046	NA	NA	NA	PCT>100	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
98	934446	NA	NA	NA	NCCT KUB: RIGHT PYELONEPHRITIS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
99	903486	NA	NA	NA	PCT 48	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
100	931917	NA	NA	NA	-	70	20	80	18	98	1226	117	-	-	-	-	27	-	-	N	Y	N	N	N	N	N	Y
101	929572	NEG	NEG	NEG	-	110	20	80	62	98	698	109	-	-	-	-	24	-	-	N	Y	N	N	N	N	N	N
102	935412	NA	NA	NA	-	140	10	90	15	79	88	98	-	-	-	-	-	POSITIVE	-	N	N	N	N	N	N	N	N
103	936136	NA	NA	NA	PCT 0.12,FUNGITELLPOSITIVE	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
104	934749	NA	NA	NA	-	200	10	90	15	101	106	112	-	-	-	-	5.5	POSITIVE	-	N	N	N	N	N	N	Y	N
105	933086	NEG	NEG	NEG	-	2	0	0	82	109	62	112	-	-	-	-	3.7	-	POSITIVE	Y	N	N	N	Y	N	N	N
106	935006	NEG	NEG	NEG	2D ECHO:GHK EF 42%	30	10	30	77	110	40	122	-	-	-	-	4	-	-	N	N	N	N	N	N	N	N
107	937358	NA	NA	NA	PCT >100	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
108	936387	NA	NA	NA	-	20	10	90	48	78	79	112	-	-	-	-	11	POSITIVE	-	N	N	N	N	N	N	Y	N
109	938278	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
110	630907	NA	NA	NA	-	40	10	90	58	210	140	113	-	-	-	-	12	-	-	N	N	N	N	N	N	N	N
111	939085	NEG	NEG	NEG	-	2	0	0	102	123	68	148	-	-	-	-	4.37	-	NEGATIVE	N	N	N	N	N	N	N	N
112	942598	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	Y	N
113	940763	NA	NA	NA	NCCT KUB: LEFT GROSS HYDROURERONEPHROSIS, PCT 4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
114	673574	NA	NA	NA	-	2	0	0	88	207	20	120	-	-	-	-	1.6	-	-	N	N	N	N	N	Y	N	N
115	940295	POSITIVE	NEG	NEG	-	2	0	0	65	125	88	90	-	-	-	-	6.8	-	POSITIVE	N	N	N	N	N	N	N	N
116	942461	NA	NA	NA	-	90	10	90	38	87	384	106	-	-	-	-	43	POSITIVE	-	Y	N	N	N	N	N	Y	N
117	934668	NA	NA	NA	PCT 3	30	60	40	87	234	115	109	-	-	-	-	6.3	POSITIVE	NEGATIVE	N	Y	N	N	N	N	N	Y
118	945392	NA	NA	NA	-	2	0	0	99	142	27	110	-	-	-	-	2.3	-	-	N	N	N	N	N	Y	N	N
119	698844	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
120	75711	NEG	NEG	NEG	PCT 44	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
121	942598	NEG	NEG	NEG	-	160	10	80	34	90	115	129	-	-	-	-	28	-	-	N	N	N	N	N	N	Y	N
122	942631	NEG	NEG	NEG	-	60	10	60	4	96	620	112	-	-	-	-	88	POSITIVE	-	N	Y	N	N	N	N	N	N
123	949182	NEG	NEG	NEG	-	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	NA	N	N	N
124	949989	NA	NA	NA	-	400	90	10	9	96	298	138	-	-	-	-	14	-	-	N	Y	N	N	N	POST EVD	N	N
125	947989	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
126	946169	NA	NA	NA	-	35	20	80	43	98	92	108	-	-	-	-	18	-	-	N	N	N	N	N	N	Y	N
127	951754	NEG	NEG	NEG	PCT NORMAL	2	0	0	115	140	42	128	-	-	-	-	2.3	-	-	N	N	N	N	N	Y	N	N

S.NO	MRD NO	MRI_RING ENHANCING LESIONS	MRI_MENINGIOENCEPHALITIS/ ENCEPHALITIS	MRI HYDROCEPHALUS	MRI_DIFFUSE CERBRAL OEDEMA	MRI NORMAL	MRI_OTHERS	EEG (if needed)	TUBERCULOUS MENINGITIS	2.VIRAL	3.DENGUE ENCEPHALOPATHY/ ENCEPHALITIS	4.PYOGENIC	5.MALARIA	6.SCRUB RELATED ENCEPHALOPATHY	7.SEPTIC ENCEPHALOPATHY	POST COVID ENCEPHALOPATHY	LEPTOSPIRA INDUCED ENCEPHALOPATHY	OTHERS	TREATMENT GIVEN	OUTCOME 1. RECOVERED, 2. DAMA,
86	923746	NA	NA	NA	NA	NA	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
87	922325	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT,STEROIDS	1
88	924790	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/INOTROPES/STEROIDS	2
89	923505	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	STEROID,ATT,ANTIEPILEPTICS	1
90	923819	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT,STEROIDS	1
91	925545	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ATIBIOTICS,INOTROPES	1
92	926731	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/ANTIEPILEPTIC	2
93	928138	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS/INOTROPES	1
94	527028	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ INOTROPES	3
95	927159	N	Y	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ANTIBIOTICS,ATT,STEROIDS	2
96	920342	N	N	N	N	N	VENTRICULITIS	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/IV ANTIBIOTICS/VENTILATORY SUPPORT	3
97	934046	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS	2
98	934446	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	2
99	903486	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/INOTROPES/SUPPORTIVE	1
100	931917	N	Y	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	IV ANTIBIOTICS/ATT/STEROIDS/ANTIEPILEPTICS	2
101	929572	Y	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
102	935412	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
103	936136	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ANTIFUNGALS/SUPPORTIVE	1
104	934749	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
105	933086	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	ANTIVIRAL/ANTIBACTERIALS	3
106	935006	N	N	N	N	Y	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIBIOTIC/ANTIVIRAL/ANTIOEDEMA	2
107	937358	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/IV STEROIDS	1
108	936387	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
109	938278	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTBIOTICS/INOTROPES/PERITONEALDIALYSIS	3
110	630907	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
111	939085	N	Y	N	N	N	S/O HERPES ENCEPHALITIS	-	N	Y	N	N	N	N	N	N	N	N	ANTIVIRAL/ANTIBACTERIALS	1
112	942598	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
113	940763	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
114	673574	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTIS/SUPPORTIVE	2
115	940295	N	Y	N	N	N	S/O HERPES ENCEPHALITIS	DIFFUSE ENCEPHALOPATHY	N	Y	N	N	N	N	N	N	N	N	IV ACYCLOVIR,IV ANTIBIOTICS,IV STEROIDS	3
116	942461	Y	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/EVD	2
117	934668	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/ANTIEPILEPTIC	1
118	945392	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ATT/STEROIDS	2
119	698844	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS/INOTROPES	3
120	75711	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS	1
121	942598	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
122	942631	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/EVD	2
123	949182	N	N	N	N	N	NA	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIBIOTICS/ANTIVIRALS/NEKSIUM INFUSION	3
124	949989	N	N	N	N	N	-	-	N	N	N	Y	N	N	N	N	N	N	IV ANTIBIOTICS/RIGHT VP SHUNT	1
125	947989	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/PERITONEAL DIALYSIS/INOTROPES	3
126	946169	Y	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	MODIFIED ATT/ IV ANTIBIOTICS/STEROIDSMANNITOL	2
127	951754	N	N	N	N	Y	-	-	N	N	N	N	N	N	N	Y	N	N	STEROID PULSE/IV ANTIBIOTICS/CLEXANE	1

S.NO	MRD NO	DOA	AGE	SEX	RELIGION	OCCUPATION	CHIEF COMPLAINT S FEVER	ALTERED SENSORIUM	HEADACHE	VERTIGO	BLURRING VISION	ABNORMAL BODY MOVEMENT	WEAKNESS IN ANY PART OF	VOMITING	OTHERS	PAST MEDICAL ILLNESS HISTORY			FAMILY HISTORY	PERSONAL HISTORY				SBP	DBP	TEMP	PULSE RATE	RR	PALOR	GCS	CLUBBING	ICTERUS	EDEMA	PETECHIAE / PURPURA
																TYPE 2 DM	HYPERTENSION	OTHERS		ALCOHOL	SMOKING	SUBSTANCE ABUSE	NONE											
128	954633	14/8/20	57	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	Y	-	-	-	80	60	100	143	28	Y	6	-	-	-	-
129	505893	15/8/20	72	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	SHORTNESSOF BREATH	-	-	-	NIL	Y	-	-	-	100	60	101	78	18	-	12	-	-	-	-
130	954159	16/8/20	22	M	SIKH	LABOUR	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	10	60	100	102	18	-	13	-	-	-	-
131	865661	19/8/20	74	M	SIKH	LABOUR	Y	Y	N	N	N	N	N	N	-	-	-	CA STOMACH	NIL	-	-	-	Y	80	60	98.2	78	19	-	12	-	-	-	-
132	956877	19/8/20	65	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	JAUNDICE	-	Y	-	NIL	-	-	-	Y	110	70	101	113	16	-	12	-	-	-	-
133	68410	20/8/20	74	M	HINDU	DEPENDENT	Y	Y	N	N	N	Y	N	Y	-	Y	-	CAD/PVD	NIL	-	Y	-	-	100	60	101	124	18	-	10	-	-	-	-
134	423960	20/8/20	69	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	LOOSE STOOL/SHORTNESS OF BREATH	Y	-	-	NIL	-	-	-	Y	110	70	101	128	32	-	10	-	-	-	-
135	955006	20/8/20	26	M	HINDU	SELF EMPLOYED	Y	Y	N	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	110	70	99	86	17	Y	12	-	-	-	-
136	948783	24/8/20	45	M	SIKH	SELF EMPLOYED	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	120	60	100	102	16	-	6	-	-	-	-
137	931556	26/8/20	46	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	Y	-	-	-	130	70	102	128	16	-	9	-	-	-	-
138	958070	29/8/20	28	M	HINDU	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	VISUAL HALLUCINATIONS	-	-	-	NIL	-	Y	Y	-	100	60	99	86	18	Y	11	-	-	-	-
139	185886	2-9-20	71	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	LOOSE STOOLS	-	Y	OLD CVA/CAD	NIL	-	-	-	Y	110	70	101	98	18	-	12	-	-	-	-
140	835267	2-9-20	62	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	LEFT LEG SWELLING	-	-	HPOTHYROID/OA/DVT	NIL	-	-	-	Y	80	60	101	110	20	Y	10	-	-	-	-
141	807316	5-9-20	65	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	Y	N	-	-	-	-	NIL	-	-	-	Y	110	60	97.3	76	20	-	4	-	-	-	-
142	963531	8-9-20	51	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	100	70	100	90	18	-	12	-	-	-	-
143	964569	18/9/20	51	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	SHORTNESS OF BREATH	Y	-	-	NIL	-	-	-	Y	100	60	100	121	18	Y	11	-	-	-	-
144	913445	11-9-20	64	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	PAINABDOMEN	-	-	CAD	NIL	-	-	-	Y	90	70	102	115	18	-	12	-	-	Y	-
145	962550	12-9-20	49	F	MUSLIM	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	120	60	100	92	21	-	12	-	-	-	-
146	965329	14/9/20	56	M	SIKH	BUSINESS	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	100	70	101	98	18	-	11	-	-	-	-
147	965799	16/9/20	25	F	HINDU	STUDENT	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	70	60	101	123	20	Y	12	-	-	-	-
148	965227	18/9/20	19	M	HINDU	STUDENT	Y	Y	N	N	N	Y	N	N	HEMOPTYSIS	-	-	-	NIL	-	-	-	Y	90	60	99	77	16	-	9	-	-	-	Y
149	965573	21/9/20	59	M	HINDU	SERVICE	Y	Y	N	N	N	N	N	N	-	-	Y	-	DENGUE IN SON	-	-	-	Y	110	70	102	132	18	-	11	-	-	-	-
150	970423	28/9/20	60	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	120	60	102	129	20	-	12	-	-	-	-
151	873551	28/9/20	52	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	N	-	-	Y	BRONCHIALASTHMA/HYPOTHYROID	NIL	-	-	-	Y	110	70	98	86	21	-	12	-	-	-	Y
152	974449	1-10-20	34	M	SIKH	SERVICE	Y	Y	N	N	N	N	N	N	-	-	-	POSTCOVID	NIL	-	-	-	Y	70	60	100	124	22	-	7	-	-	-	-
153	968012	1-10-20	18	M	HINDU	STUDENT	Y	Y	N	N	N	N	N	N	BDOMINAL DISTENSION	-	-	-	NIL	-	-	-	Y	100	60	100	123	23	Y	6	-	-	-	-
154	972143	4-10-20	36	M	SIKH	SERVICE	Y	Y	N	N	N	N	N	N	-	-	-	SEIZURE DISORDER	NIL	-	-	-	Y	110	70	99.8	112	18	-	12	-	-	-	-
155	982279	9-10-20	42	M	HINDU	AGRICULTURE	Y	Y	N	N	N	N	N	N	-	-	-	CAD	NIL	Y	-	Y	-	110	70	99	110	20	-	10	-	-	-	-
156	973045	10-10-20	38	M	MUSLIM	SERVICE	Y	Y	Y	N	Y	N	N	N	-	-	-	-	NIL	-	-	-	Y	80	50	102	124	24	-	4	-	-	-	-
157	974492	10-10-20	76	M	HINDU	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	Y	-	CAD	NIL	-	Y	-	-	100	60	100	98	26	-	9	Y	-	-	-
158	975941	14/10/20	51	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	N	JAUNDICE	-	-	-	DENGUE TWICE BEFORE	-	-	-	Y	100	50	102	156	18	-	10	-	Y	-	Y
159	976272	15/10/20	52	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	N	SHORTNESS OF BREATH	-	-	-	NIL	Y	-	-	Y	110	60	99.8	109	20	-	8	-	-	Y	-
160	976745	16/10/20	86	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	Y	-	Y	-	-	NIL	-	-	-	Y	90	60	100	118	18	-	8	-	-	Y	-
161	977161	17/10/20	72	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	JAUNDICE	Y	-	-	NIL	-	-	-	Y	110	60	98	112	18	-	12	-	Y	Y	-
162	977324	18/10/20	42	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	Y	-	-	-	-	DENGUE IN SON	-	-	-	Y	80	50	103	156	22	-	7	-	-	-	-
163	968666	21/10.20	85	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	CAD	NIL	-	-	-	Y	100	60	100	118	18	-	12	-	-	-	-
164	978904	22/10/21	45	M	HINDU	GOVT JOB	Y	Y	N	N	N	Y	N	N	-	Y	-	-	NIL	-	-	-	Y	110	80	102	112	18	-	11	-	-	-	-

S.NO	MRD NO	LYMPHADENOPATHY	CYANOSIS	CENTRAL NERVOUS SYSTEM	CVS	RS	P/A	HB	TLC	DLC	PLT	ESR	PBF MALARIA	PTI-INR	UREA	CREATININE	NA	K	S. BILIRUBIN	TOTAL PROTEIN	ALBUMIN	ALP	SGOT	SGPT	CALCIUM	ABG	CXR	ECG	BLOOD C/S/URINE C/S	Malaria Antigen
128	954633	-	-	HMF ALTERED, B/LPLANTAR EXTENSOR	NAD	B/L DIFFUSE SPASM	NAD	7.6	28.9	87/7/5	129	34	NEG	1.78	178	8.7	143	4	0.67	5.8	3.14	102	56	87	8	ACIDOSIS	B/L DIFFUSE INFILTRATES	ABNORMAL	URINE C/S:E.COLI	NA
129	505893	-	-	HMF ALTERED	NAD	RIGHT LOWER ZONES CREPTS	NAD	10.2	6.3	82/10/8	189	45	NEG	1.09	40	0.5	140	4	1.23	5.7	2.5	286	56	98	7.3	ACIDOSIS	RIGHT LOWER ZONE PATCH	NORMAL	1	NA
130	954159	-	-	HMF ALTERED	NAD	NAD	NAD	13.8	8.911.	76/13/10/2	256	82	NEG	1.23	30	0.68	129	3.72	1	8	4.18	88	37	73	8.2	NORMAL	NORMAL	NORMAL	1	NEG
131	865661	-	-	HMF ALTERED, B/LPLANTAR FLEXOR	NAD	NAD	NAD	11.7	15.1	84/12/4	120	35	NEG	1.56	129	3	138	4	0.76	6	2.33	367	73	8.2	9	NORMAL	NORMAL	NORMAL	URINEC/S:CANDIDA	NEG
132	956877	-	-	HMF ALTERED	NAD	NAD	NAD	12.7	20.7	75/15/10	205	54	NEG	1.17	89	2.12	146	4.76	1.87	6	2.58	159	94	116	9	NORMAL	NORMAL	NORMAL	1	NEG
133	68410	-	-	HMF ALTERED, B/L PLANTAR EXTENSOR	ABNORMAL	NAD	NAD	7.6	27.8	76/18/6	470	35	NEG	1.09	126	4.99	137	4.4	0.4	4.4	1.84	42	12	20	7.9	ACIDOSIS	B/L PLEURAL EFFUSION	NORMAL	WOUND SWAB:PSEUDOMONAS	NEG
134	423960	-	-	HMF ALTERED	NAD	B/L DIFFUSE CREPTS	NAD	8.9	23.6	92/6/2	187	42	NEG	0.97	52	1.2	134	3.8	0.36	6.2	3.14	167	67	98	8	TYPE 1 RF	ARDS	ABNORMAL	URINE C/S:E.COLI	NEG
135	955006	-	-	HMF ALTERED	NAD	NAD	NAD	9.6	4.8	68/22/8/2	286	32	NEG	1.32	30	0.6	129	4.3	0.92	7.4	3.46	103	61	36	8.2	NORMAL	NORMAL	NORMAL	1	NA
136	948783	-	-	ON VENTILATORY SUPPORT, B/L PLANATAR MUTE,RIGHT HEMIPARESIS	NAD	B/L AE REDUCED	NAD	8.7	9.6	90/8/2/1	340	66	NEG	1.78	20	0.53	135	4.2	0.45	5.12	2.91	88	75	67	7.8	METABOLIC AND LACTIC ACIDOSIS	PATCH OF PNEUMONITIS	SINUS TACHYCARDIA	ET C/S: E.COLI	NA
137	931556	-	-	ANISOCORIA,RIGHT BABINSKI,NECK RIGIDITY,CLONUS	NAD	NAD	NAD	7.5	7.7	84/12/3/0.3	306	73	NEG	1.21	27	0.47	131	4	0.33	6.4	3.13	96	23	20	7.9	ABNORMAL	NORMAL	NORMAL	1	NA
138	958070	Y	-	HMF ALTERED,MENINGEAL SIGNS	NAD	NAD	NAD	13.4	11.7	90/8/2	200	16	NEG	1.15	30	0.56	141	4	0.8	6.9	3.6	88	77	103	8.2	NORMAL	NORMAL	NORMAL	1	NEG
139	185886	-	-	HMF ALTERED	NAD	B/L LOWER AREAS CREPTS	NAD	11.9	8.9	79/10/10	90	22	NEG	1.24	30	0.6	135	4.2	1.11	7.8	4	93	24	19	9.2	NORMAL	NORMAL	NORMAL	1	NEG
140	835267	-	-	HMF ALTERED	NAD	LEFT SIDED COARSE CREPTS	NAD	6.7	8.8	70/28/2	235	56	NEG	1.13	40	1.03	142	3.5	0.45	5.6	2.7	180	56	78	8.1	ACIDOSIS	LEFT SIDED PATCH	NORMAL	SWAB C/S: MRSA; URINE C/S: E.COLI	NEG
141	807316	-	-	B/L LL WEAKNESS LEFT>RIGHT,BL PLANTAR UPGOING,HMF ALTERED	NAD	NAD	NAD	7	11.6	82/20/7/3	421	23	NEG	1.2	60	0.49	125	3.8	0.8	6.2	3.02	111	25	82	8.6	NORMAL	LEFT MZ OPACITY	NORMAL	ET C/S:KLEBSIELLA	NEG
142	963531	-	-	HMF ALTERED	NAD	NAD	NAD	12.9	11.8	71/24/5/4	118	34	NEG	1.08	86	1.27	140	5	3.13	5.5	2.79	247	72	81	8.1	NORMAL	PERI HILAR OPACITIES	ABNORMAL	1	NEG
143	964569	-	-	HMF ALTERED, NO ENINGEAL SIGNS, B/LPLANTAR WITHDRAWL	NAD	NAD	NAD	7.7	26	91/7/1	182	37	NA	1.32	98	4.52	136	4	1.25	5.8	2.34	189	69	50	7.8	LACTIC ACIDOSIS	B/L CP ANGLES BLUNT	NORMAL	PUS C/S: ACINTEOBACTER/ MRSA	NA
144	913445	-	-	HMF ALTERED	NAD	NAD	NAD	7.5	15.4	70/22/8	489	24	NEG	1.35	67	2.91	129	5.4	0.49	6.9	3.25	86	16	9	7.1	ACIDOSIS	NORMAL	NORMAL	URINE C/S:E.COLI	NEG
145	962550	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	9.9	10.5	79/13/8/2	240	46	NEG	1.18	45	0.49	122	3.43	0.6	7.1	3.7	71	55	57	9.3	NORMAL	NORMAL	NORMAL	1	NEG
146	965329	-	-	HMF ALTERED	NAD	NAD	NAD	9.1	13.5	82/12/8	351	31	NEG	1.09	41	0.32	131	4	0.52	6.9	3.14	78	367	241	8.1	NORMAL	NORMAL	NORMAL	1	NEG
147	965799	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	8.2	8	78/18/2.7	177	15	NEG	1.18	104	1.4	155	4.51	0.6	6.8	3.14	127	70	40	8.9	LACTIC ACIDOSIS	MILIARY SHADOWS	NORMAL	1	NEG
148	965227	-	-	HMF ALTERED,INTUBATED,MOVING ALL 4 LIMBS,PERSISTENT SEIZURES	NAD	B/L DIFFUSE CONDUCTED SOUNDS	NAD	7.8	11.8	85/15/1	45	56	NEG	1.45	46	0.4	134	3.7	3.5	6	3.26	100	789	546	7	MIXED ACIDOSIS	ARDS	ABNORMAL	1	NEG
149	965573	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	B/L DECREASED AIR ENTRY	HEPATOSPLENOMEGALY	10.5	15.5	54/37/6	48	21	NEG	2.97	114	4.86	136	4.8	6.36	6.9	2.28	441	345	303	7.7	ACIDOSIS	B/LPLEURAL EFFUSION	ABNORMAL	1	NEG
150	970423	-	-	HMF ALTERED	NAD	B/L LZ AIR ENTRY REDUCED	NAD	11.6	9.5	78/20/2	103	65	NEG	1.01	38	1.31	140	4	0.27	7.1	3.25	213	241	166	8.4	NORMAL	B/LPLEURAL EFFUSION	NORMAL	1	NEG
151	873551	-	-	HMF ALTERED	NAD	NAD	NAD	9.7	9	68/20/12	397	36	NA	1.47	18	0.69	135	4.1	0.37	6.5	3.66	62	137	70	7	NORMAL	B/L CP ANGLES BLUNT	NORMAL	1	NEG
152	974449	-	-	HMF ALTERED	NAD	B/L COARSE CREPTS	NAD	12.1	37.4	88/10/2	90	80	NA	1.78	108	1.63	140	4.8	0.6	7.6	5	125	25	29	10.3	LACTIC ACIDOSIS	B/LARDS	ABNORMAL	BLOOD C/S : PSUEDOMONAS	NEG
153	968012	-	-	INTUBATED, MOVING ALL 4 LIMBS, B/L PLANTAR EXTENSOR	NAD	NAD	DISTENDED,HEPATOSPLENOMEGALY	8.5	10	78/12/8/2	70	56	NEG	1.43	40	0.7	134	4	0.9	6	3	97	870	435	8.9	METABOLIC ACIDOSIS	RIGHT LZ HAZINESS	ABNORMAL	1	NEG
154	972143	-	-	HMF ALTERED	NAD	NAD	NAD	9.5	16.7	83/12/8	274	18	NEG	1.37	10	0.5	144	4.5	0.28	5.4	3.6	111	92	89	9.1	NORMAL	NORMAL	NORMAL	BLOOD C/S:BURKHOLDERIA	NEG
155	982279	-	-	HMF ALTERED,NECK RIGIDITY PRESENT	NAD	NAD	NAD	8.6	16.7	93/6/1/0	1268	87	NEG	1.23	45	0.72	141	4.5	0.15	6.4	3.37	98	36	78	9.7	NORMAL	NORMAL	ABNORMAL	1	NEG
156	973045	-	-	DECORTICATE RIGIDITY,INTUBATED,B/L PLANTAR MUTE	NAD	NAD	NAD	8.5	1.2	77/12/13	22	82	NEG	1.54	32	1.54	151	4.22	0.8	5.6	2.66	187	476	194	8	METABOLIC AND LACTIC ACIDOSIS	B/L PLEURAL EFFUSION	ABNORMAL	1	NEG
157	974492	-	-	HMF ALTERED,RIGHT PLANTAR EXTENSOR	NAD	B/L AE REDUCED,SPASM	NAD	9.8	38	94/1/4	22	21	NEG	1.09	89	2.96	130	4.65	0.73	5.3	2.31	196	34	46	8.1	ACIDOSIS	B/L PLEURAL EFFUSION	ABNORMAL	1	NEG
158	975941	-	-	HMF ALTERED	NAD	B/LAE REDUCED	HEPATOMEGALY	15.3	28.1	70/20/10	50	54	NEG	1.9	35	1.12	143	4	8.6	6	3.77	268	7780	3975	8.3	ACIDOSIS	B/LPLEURAL EFFUSION	ABNORMAL	1	NEG
159	976272	-	-	HMF ALTERED, B/L PLANTAR EXTENSOR	NAD	B/L REDUCED AIR ENTRY	NAD	9.1	13.2	54/18/12	484	35	NEG	1.9	42	2.3	136	5	0.41	5.1	2.33	70	29	14	7.9	ACIDOSIS	B/L PLEURAL EFFUSION	NORMAL	1	NEG
160	976745	-	-	HMF ALTERED	NAD	B/L DIFFUSE CREPTS	NAD	8.9	18.1	90/6/4	226	87	NEG	1.09	127	4.1	138	4.7	1.21	5.9	2.89	160	117	243	9	ACIDOSIS	NORMAL	NORMAL	BLOOD C/S: ELIZABETHKINGIA MENINGOSEPTICA	NEG
161	977161	-	-	HMF ALTERED	NAD	NAD	NAD	10.5	9.2	76/11/10/	50	22	NEG	1.41	127	3.44	143	7	1.81	6	2.89	218	862	1387	8.7	ACIDOSIS	NORMAL	NORMAL	1	NEG
162	977324	-	-	HMF ALTERED,B/L PLANTAR MUTE	NAD	B/L AE REDUCED	NAD	7.8	13.4	71/17/10	70	34	NEG	1.71	104	5.1	137	3.6	2.34	5.9	3.1	266	453	254	8	ACIDOSIS	ARDS	ABNORMAL	1	NEG
163	968666	-	-	HMF ALTERED	ABNORMAL	B/L PERI HILAR PROMINENCE	NAD	11.1	31.7	92/6/2	223	43	NEG	1.17	129	2.01	144	4.4	1.05	6.3	2.79	106	86	40	8	ACIDOSIS	B/LPERI HILAR PROMINENCE	ABNORMAL	APUTUM C/S:KLEBSIELLA; URINE: E.COLI	NEG
164	978904	-	-	HMF ALTERED, MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	12.4	13.4	83/18/2	120	78	NEG	1.32	22	0.3	135	3.4	0.89	7.2	3.78	88	29	67	8.9	NORMAL	NORMAL	NORMAL	BLOOD:KLEBSIELLA / URINE : E.COLI	NEG

S.NO	MRD NO	Leptospira	Dengue	Scrub Typhus	Other Investigations	CSF CYTOLOGY_TLC	NEUTROPHILS	LYMPHOCYTES	GLUCOSE	S. GLUCOSE	PROTEIN	CHLORIDE	GRAM STAIN	INDIA INK PREPARATION	BACTERIAL ANTIGEN	CULTURE SENSITIVITY	ADA	CBNAAT	HSV PCR	CT HEAD_FOCAL HYPODENSITIES	CT HEAD HYDROCEPHALUS	CT HEAD_DIFFUSE CEREBRAL OEDEMA	CT HEAD INFARCT	CT HEAD OTHERS	CT HEAD NORMAL	MRI LEPTOMENINGITIS	MRI VASCULITIC INFARCT
128	954633	NA	NA	NA	NCCT KUB : EMPHYSEMATOUS CYSTITIS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
129	505893	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
130	954159	NEG	NEG	NEG	-	70	25	75	11	77	144	106	-	-	-	-	12	-	-	N	N	N	N	N	N	Y	N
131	865661	NEG	NEG	NEG	PCT -4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
132	956877	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
133	68410	NEG	NEG	NEG	PCT 1.58	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
134	423960	NA	NA	NA	PCT 2.2	2	0	0	115	165	35	120	-	-	-	-	3.1	-	-	N	N	N	N	N	N	N	N
135	955006	NA	NA	NA	-	2	0	0	11	112	125	113	-	-	-	-	14	POSITIVE	-	N	N	N	N	N	N	Y	N
136	948783	NA	NA	NA	-	20	10	90	36	78	104	113	-	-	-	-	6.09	-	POSITIVE	N	N	N	N	N	N	N	N
137	931556	NA	NA	-	-	15	10	90	54	163	87	116	-	-	-	-	92	POSITIVE	-	N	Y	N	N	N	N	N	Y
138	958070	POSITIVE	NEG	NEG	-	30	10	90	40	109	278	115	-	-	-	-	20	POSITIVE	-	N	N	N	N	N	N	Y	Y
139	185886	NEG	NEG	POSITIVE	2D ECHO: NORMAL	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	DIFFUSE CEREBRAL ATROPHY	N	N	N
140	835267	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
141	807316	NA	NA	NA	-	10	5	95	71	196	800	112	-	-	-	-	29	POSITIVE	-	N	N	N	N	N	N	Y	N
142	963531	NEG	NEG	POSITIVE	-	20	20	80	57	110	115	128	-	-	-	-	9.04	-	-	N	N	N	N	N	N	N	N
143	964569	NA	NA	NA	PCT-6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
144	913445	NA	NA	NA	PCT 11	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
145	962550	NA	NA	NA	-	20	20	80	69	94	114	103	-	-	-	-	24	-	-	N	N	N	N	N	N	N	N
146	965329	NA	NA	NA	-	15	10	90	72	98	58	88	-	-	-	-	1.86	-	-	N	N	N	N	N	Y	N	N
147	965799	NA	NA	NA	-	200	10	90	80	98	432	130	-	-	-	-	43	POSITIVE	-	N	N	N	N	N	N	Y	N
148	965227	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
149	965573	POSITIVE	POSITIVE	POSITIVE	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
150	970423	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
151	873551	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
152	974449	NEG	NEG	NEG	PCT-5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	NA	N	N	N
153	968012	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
154	972143	NEG	NEG	NEG	PCT 4.3	10	10	90	75	102	58	102	-	-	-	-	2.86	-	-	N	N	N	N	N	N	N	N
155	982279	NEG	NEG	NEG	-	1600	90	10	67	-	67/108	76	133	-	-	-	-	-	-	N	N	N	N	N	N	1	N
156	973045	NEG	POSITIVE	NEG	-	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	Y	N	N	N	N	N
157	974492	NEG	NEG	NEG	FUNGITELL 523/GALACTOMANNAN 2.3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
158	975941	NEG	POSITIVE	NEG	PCT 0.9,VIRAL MARKERS NEGATIVE,AMMONIA 127	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N/L CEREBRAL CALCIFICATIONS	N	N	N
159	976272	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	MULTIPLE SMALL HEMMORHAGIC FOCI	N	N	N
160	976745	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
161	977161	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
162	977324	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
163	968666	NEG	NEG	NEG	PCT 27	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	Y
164	978904	NEG	NEG	NEG	PCT1.07	2	0	0	83	99	38	131	-	-	-	-	1.45	-	POSITIVE	N	N	N	N	N	N	N	N

S.NO	MRD NO	MRI_RING ENHANCING LESIONS	MRI_MENINGIOENCEPHALITIS/ ENCEPHALITIS	MRI HYDROCEPHALUS	MRI_DIFFUSE CERBRAL OEDEMA	MRI NORMAL	MRI_OTHERS	EEG (if needed)	TUBERCULOUS MENINGITIS	2.VIRAL	3.DENGUE ENCEPHALOPATHY/ ENCEPHALITIS	4.PYOGENIC	5.MALARIA	6.SCRUB RELATED ENCEPHALOPATHY	7.SEPTIC ENCEPHALOPATHY	POST COVID ENCEPHALOPATHY	LEPTOSPIRA INDUCED ENCEPHALOPATHY	OTHERS	TREATMENT GIVEN	OUTCOME 1. RECOVERED, 2. DAMA,
128	954633	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS/VENTILATOR/	3
129	505893	N	N	N	N	N	-	MODERATE DIFFUSE ENCEPHALOPATHY	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/IV STEROIDS	1
130	954159	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
131	865661	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IVANTIBIOTICS	1
132	956877	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
133	68410	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/INOTROPES/STEROIDS	2
134	423960	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/IV STROIDS/CLEXANE	3
135	955006	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
136	948783	N	Y	N	N	N	HYPERINTENSITIES	MODERATE DIFFUSE ENCEPHALOPATHY	N	Y	N	N	N	N	N	N	N	N	IV ACYCLOVIR/IV ANTIBIOTICS/STEROIDS	1
137	931556	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT /STEROIDS	1
138	958070	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
139	185886	N	N	N	N	N	-	-	N	N	N	N	N	Y	N	N	N	N	IV DOXY,ANTIEPILEPTIC,	1
140	835267	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROID/INOTROPES	2
141	807316	N	Y	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/CYCLOPHOSPHAMIDE	1
142	963531	N	N	N	N	N	HEMMORHAGE IN LEFT POSTERIOR PARIETAL REGION	-	N	N	N	N	N	Y	N	N	N	N	IV DOXY,IV ANTIEPILEPTICS/STEROIDS	1
143	964569	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS	1
144	913445	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IB ANTIBIOTICS/SUPPORTIVE	1
145	962550	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEOIDS	1
146	965329	N	N	N	N	N	Y	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS/IV FLUIDS/SUPPORTIVE	1
147	965799	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/VENTILATOR	3
148	965227	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,IV STEROIDS,VENTILATORY SUPPORT,INOTROOPES	3
149	965573	N	Y	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,VENTILATORY SUPPORT, INOTROPES	3
150	970423	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV MANNITOL/STEROIDS/FLUIDS	1
151	873551	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,STEROIDS,ANTIHYPERTENSIVE DRUGS	1
152	974449	N	N	N	N	NA	-	MODERATE DIFFUSEENCEPHALOPATHY	N	N	N	N	N	N	Y	N	N	N	IV NATIBIOTICS,STEROIDS, ECMO	3
153	968012	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV FLUIDS,IV ANTIBIOTICS,INOTROPES,VENTILATORY SUPPORT	3
154	972143	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS, IV FLUIDS,INOTROPES	3
155	982279	N	N	N	N	N	-	-	N	N	N	Y	N	N	N	N	N	N	IV ANTIBIOTICS,STEROIDS	1
156	973045	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,ANTI OEDEMA,INOTROPES	3
157	974492	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIFUNGAL/ANTIBIOTIV/INOTROPES/SUPPORTIVE	1
158	975941	N	N	N	N	N	EXTENSIVE GLIOSIS IN B/L PAIETOFRONTAL LOBES(PRIOR INSULT)	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,FLUIDS,INOTROPES,ANTI HEPATIC COMA	2
159	976272	N	Y	N	N	N	HEMMORHAGIC ENCEPHALITIS	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS/TRACHEOSTOMY/ANTI OEDEMA/SUPPIRTIVE	1
160	976745	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBOTOICS/VENTILATOR/INOTROPES	3
161	977161	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV FLUIDS,ANTI HEPATIC COMA	1
162	977324	N	N	N	N	Y	-	-	N	N	Y	N	N	N	N	N	N	N	IV FLUIDS/BLOOD/IV ANTIBIOTICS/STEROIDS/CRRT/VENTILATOR	3
163	968666	N	N	N	N	N	SEPTIC INFARCTS	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ECOSPRIN/SUPPORTIVE	1
164	978904	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRAL/ANTIEPILEPTICS/IV ANTIBIOTICS	3

S.NO	MRD NO	DOA	AGE	SEX	RELIGION	OCCUPATION	CHIEF COMPLAINTS FEVER	ALTERED SENSORIUM	HEADACHE	VERTIGO	BLURRING VISION	ABNORMAL BODY MOVEMENT	WEAKNESS IN ANY PART OF	VOMITING	OTHERS	PAST MEDICAL ILLNESS HISTORY			FAMILY HISTORY	PERSONAL HISTORY				SBP	DBP	TEMP	PULSE RATE	RR	PALOR	GCS	CLUBBING	ICTERUS	EDEMA	PETECHIAE / PURPURA
																TYPE 2 DM	HYPERTENSION	OTHERS		ALCOHOL	SMOKING	SUBSTANCE ABUSE	NONE											
165	975285	22/10/20	41	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	99	86	22	-	12	-	-	-	-
166	979358	24/10/20	65	M	HINDU	SERVICE	Y	Y	N	N	N	N	N	Y	-	Y	-	-	NIL	-	-	-	Y	100	50	102	133	16	-	11	-	-	-	Y
167	977256	27/10/20	39	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	Y	-	-	-	100	60	100	110	20	-	12	-	-	-	-
168	977162	27/10/20	73	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	DENGUEIN WIFE	-	-	-	Y	110	70	101	113	22	-	10	-	-	-	-
169	980309	28/10/20	82	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	CAD	NIL	-	-	-	Y	110	70	98	86	21	-	12	-	-	-	-
170	978940	28/10/20	60	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	Y	-	Y	-	-	NIL	Y	-	-	-	90	50	101	110	18	-	10	-	-	-	-
171	791852	28/10/20	31	M	HINDU	SERVICE	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	Y	-	-	100	70	103	160	16	Y	7	-	Y	-	Y
172	975137	28/10/20	40	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	N	SOB	-	-	-	NIL	-	-	-	Y	60	40	99.2	48	22	Y	12	-	-	-	-
173	981400	29/10/20	48	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	PAINABDOMEN	Y	-	HBV	NIL	-	-	-	Y	100	60	98.5	112	20	-	11	-	-	-	-
174	981849	30/10/20	38	F	SIKH	DEPENDENT	Y	Y	Y	N	N	Y	N	Y	-	-	-	-	NIL	-	-	-	Y	100	60	101	1115	18	-	11	-	-	-	Y
175	980747	30/10/20	33	M	HINDU	SERVICE	Y	Y	N	N	N	Y	N	N	COUGH/RIGHT SIDED CHEST PAIN	-	-	-	NIL	Y	-	-	-	90	60	102	156	36	-	7	-	-	-	-
176	977161	2-11-20	72	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	100	60	100	80	20	-	12	-	-	-	-
177	981093	2-11-20	41	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	Y	BLEEDING P/V	Y	-	-	NIL	-	-	-	Y	110	70	101	99	28	Y	12	-	-	-	Y
178	982178	2-11-20	70	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	Y	-	NIL	-	-	-	Y	130	80	100	94	18	-	11	-	Y	-	-
179	983103	3-11-20	70	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	Y	-	NIL	-	Y	-	-	110	70	101	124	18	-	10	-	-	-	-
180	983143	3-11-20	48	F	SIKH	SERVICE	Y	Y	N	N	N	Y	N	N	-	-	-	RA	NIL	-	-	-	Y	100	60	101	122	22	-	11	-	-	-	-
181	983854	6-11-20	76	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	CAD/COPD	NIL	-	-	-	Y	90	60	100	89	18	-	12	-	-	-	-
182	980254	6-11-20	65	M	HINDU	AGRICULTURE	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	Y	-	-	-	80	50	103	135	18	Y	10	Y	-	-	Y
183	984893	8-11-20	68	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	MM	NIL	-	-	-	Y	90	60	100	108	20	Y	10	-	-	-	-
184	213430	9-11-20	49	M	SIKH	BUSINESS	Y	Y	N	N	N	N	N	Y	-	Y	Y	-	NIL	Y	-	-	-	110	70	100	123	28	-	12	-	-	-	-
185	140417	10-11-20	81	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	SOB	-	Y	-	NIL	-	-	-	Y	140	70	99.3	86	17	-	12	-	-	-	-
186	982168	11-11-20	51	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	N	-	Y	Y	-	NIL	-	-	-	Y	100	60	100	95	16	-	12	-	-	-	-
187	986592	12-11-20	91	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	Y	CHRONIC AF	NIL	Y	-	-	-	110	60	98.3	102	20	Y	11	-	-	-	-
188	1207450	14/11/20	29	F	HINDU	DEPENDENT	Y	Y	N	N	N	Y	N	N	LOOSE STOOLS	-	-	-	NIL	-	-	-	Y	110	70	101	123	22	Y	10	-	-	-	-
189	288933	15/11/20	73	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	MIXTARD INSULIN	-	-	-	Y	110	70	99	112	20	Y	12	-	-	-	-
190	987800	16/11/20	53	M	SIKH	SERVICE	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	Y	-	100	70	101	110	19	-	9	-	-	-	-
191	785234	18/11/20	27	F	HINDU	DEPENDENT	Y	Y	N	N	Y	N	N	N	-	-	-	-	NIL	-	-	-	Y	130	70	100	90	22	-	12	-	-	-	-
192	971448	20/11/20	82	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	POST COVID	NIL	-	-	-	Y	100	60	101	135	29	-	5	-	-	-	-
193	990970	23/11/20	55	M	SIKH	BUSINESS	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	100	60	101	112	20	-	10	-	-	-	-
194	990984	23/11/20	71	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	80	40	102	118	22	-	7	-	-	-	-
195	991359	24/11/20	81	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	RIGHT LOWER LIMB CELLULITIS	Y	-	-	NIL	-	-	-	Y	110	70	100	98	18	-	10	-	-	-	-
196	991446	25/11/20	53	F	SIKH	GOVT JOB	Y	Y	N	N	N	Y	N	N	-	Y	Y	-	NIL	-	-	-	Y	130	60	101	110	22	-	8	-	-	-	-
197	992248	26/11/20	30	F	SIKH	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	80	40	102	138	19	-	10	-	-	-	-
198	987795	28/11/20	25	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	130	70	100	117	16	-	12	-	-	-	-
199	993161	28/11/20	65	M	SIKH	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	Y	-	OLD CVA	NIL	Y	-	-	-	80	60	99	145	20	-	9	-	-	-	-
200	989223	28/11/20	46	F	SIKH	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	130	50	101	120	16	Y	12	-	-	-	-
201	993199	28/11/20	22	F	HINDU	STUDENT	Y	Y	N	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	110	70	101	108	22	-	11	-	-	-	-

S.NO	MRD NO	LYMPHADENOPATHY	CYANOSIS	CENTRAL NERVOUS SYSTEM	CVS	RS	P/A	HB	TLC	DLC	PLT	ESR	PBF MALARIA	PTI-INR	UREA	CREATININE	NA	K	S. BILIRUBIN	TOTAL PROTEIN	ALBUMIN	ALP	SGOT	SGPT	CALCIUM	ABG	CXR	ECG	BLOOD C/S/URINE C/S	Malaria Antigen
165	975285	-	-	CONFUSED,IRRE;EVANT TALKS	NAD	NAD	NAD	13.7	6.9	68/19/1.8	278	42	NEG	1.23	17	0.49	141	3.46	0.43	6.3	3.3	105	17	15	9	NORMAL	NORMAL	NORMAL	BLOOD:BURKHOLDERIA CEPACIA	NEG
166	979358	-	-	HMF ALTERED	NAD	B/LAE REDUCED	HEPATOSPLENOMEGALY	13.6	7.4	70/18/2	42	43	NEG	1.35	41	0.83	140	3.5	1.05	6.6	3.34	117	372	275	8.4	NORMAL	NORMAL	NORMAL	1	NEG
167	977256	-	-	HMF ALTERED	NAD	NAD	HEPATOSPLENOMEGALY	12.8	28	92/7/1	26	63	NEG	1.46	53	1.15	144	4.2	0.86	5.3	3.18	77	3629	1347	7.9	NORMAL	NORMAL	NORMAL	1	NEG
168	977162	-	-	HMF ALTERED	NAD	NAD	NAD	9.8	28.4	88/8/4	100	65	NEG	2.2	118	4.34	134	4	1.49	5.7	2.48	415	49	27	8	NORMAL	NORMAL	NORMAL	BLOOD C/S: KLEBSIELLA	NEG
169	980309	-	-	HMF ALTERED	NAD	B/L CREPTS	NAD	12.7	19.5	98/2	118	43	NA	1.56	78	2.34	138	4.9	1.98	5.7	2.67	177	67	89	9	ACIDOSIS	B/L ARDS	ABNORMAL	1	NEG
170	978940	-	-	HMF ALTERED	NAD	NAD	NAD	10	7.4	66/12/12	78	56	NEG	1.23	56	1.4	136	4.2	0.9	7	4.2	110	650	223	8	RESPIRATORY ALKALOSIS	B/L CP ANGLE BLUNTING	NORMAL	1	NEG
171	791852	-	-	INTUBATED, B/LPLANTAR EXTENSOR,B/L PUPILS SLUGGISH	ABNORMAL	B/L AE REDUCED,CONDUCTED SOUNDS	HEPATOMEGALY, ABDOMINAL DISTENSION	7.6	19	87/13/8/2	65	47	NEG	1.76	90	2.3	134	4.5	1.8	6.7	2.34	209	670	435	7.6	METABOLIC AND LACTIC ACIDOSIS	ARDS	ABNORMAL	1	NEG
172	975137	-	-	HMF ALTERED	NAD	NAD	LIVER PALPABLE	7.3	7.2	76/13/9	120	18	NEG	2	54	1.1	148	3.8	1.36	4.4	2	109	259	182	7.2	METABOLIC ACIDOSIS	ARDS	ABNORMAL	1	NEG
173	981400	-	-	HMF ALTERED	NAD	NAD	HEPATOMEGALY,DISTENDED	12	13.4	88/10/2	118	12	NEG	1.09	79	1.92	138	4	0.22	4.8	2.23	154	19	17	7.2	NORMAL	B/L PLEURAL EFFUSION	NORMAL	1	NEG
174	981849	-	-	DROWSY,RESPONSE TO PAIN,B/L PLANTAR FLEXOR,NECK RIGDITY	NAD	NAD	NAD	11	29.7	88/10/2/1	80	81	NEG	1.23	104	1.61	150	3.6	1.75	5.6	2.16	105	104	68	8.4	METABOLIC ACIDOSIS	RIGHT LZ HAZINESS	ABNORMAL	MRSA IN BLOOD	NEG
175	980747	-	-	HMF ALTERED,MOVING ALL 4 LIMBS,RIGHT PLANTAR EXTENSOR	NAD	DECREASED LEFT AIRENTRY	NAD	11.8	40	92/6/2	177	120	NEG	1.08	78	1.7	146	5	0.9	5.7	2.91	230	176	70	8.1	ACIDOSIS,TYPE 1 RF	RIGHT PLEURAL EFFUSION/ICD IN SITU	ABNORMAL	PLEURAL FLUID C/S: STREPTOCOCCUS PNEUMONIAE	NEG
176	977161	-	-	HMF ALTERED	NAD	NAD	NAD	10.7	8.5	67/13/12	88	67	NEG	1.34	113	3.44	142	4.95	1.29	5.2	2.34	84	378	84	6.7	METABOLIC ACIDOSIS	NORMAL	NORMAL	1	NEG
177	981093	-	-	HMF ALTERED,DROWSY,RESTLESS	NAD	NAD	NAD	7.6	9.7	77/13/10	26	18	NEG	1.45	96	1.11	130	6.2	2.52	5.7	3	305	3201	1101	7	HYPERKALEMIA	B/L CP ANGLE BLUNTING	ABNORMAL	1	NEG
178	982178	-	-	HMF ALTERED, B/L PLANTAR WITHDRAWL, MOVING ALL 4 LIMBS, ALL RLEFLEXES PRESENT AND 2 +	NAD	NAD	SPLENOMEGALY	14.6	6.6	78/14/10	44	46	NEG	1.19	64	1.57	128	4	0.61	6.2	2.62	44	83	91	8.2	NORMAL	NORMAL	NORMAL	1	NEG
179	983103	-	-	HMF ALTERED	NAD	B/L SPASM	NAD	12.4	18.2	92/6/2	245	25	NEG	1.09	61	1.56	147	4.7	0.71	6.6	2.86	71	82	125	7.9	NORMAL	RIGHTLZ HAZINESS	NORMAL	1	NEG
180	983143	-	-	HMF ALTRERED, B/LPLANTAR EXTENSOR	NAD	NAD	NAD	12.6	4.5	70/28/2	116	45	NEG	1.02	65	1.4	135	3.6	0.22	6.9	3.96	102	36	30	8	NORMAL	NORMAL	NORMAL	1	NEG
181	983854	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	B/L CONDUCTED SOUNDS	NAD	12.3	13.1	85/15	244	19	NEG	1.34	20	0.61	131	3.6	0.79	4	2.57	73	30	31	8.7	NORMAL	B/LDISSUSE HAZINESS	ABNORMAL	1	NEG
182	980254	-	-	HMF ALTERED,RESTLESS	NAD	NAD	PALPABLE LIVER	7.1	2.1	83/7/5/5	50	35	NEG	1.21	114	7.03	146	5	3.66	6.2	3.2	86	718	215	7.6	MIXED ACIDOSIS	B/L LZ HAZINESS	NORMAL	1	NEG
183	984893	-	-	HMF ALTERED, B/LPLANTAR MUTE	NAD	NAD	NAD	8.2	8.4	83/8/9	90	118	NEG	1.45	48	1.8	145	3.5	1.4	6.6	1.77	116	28	140	6.5	ACIDOSIS	B/L DIFFUSE INFILTRATES	NORMAL	1	NEG
184	213430	-	-	HMF ALTERED	NAD	B/L CREPTS	HEPATOMEGALY	13	12.4	82/9/10	22	21	NEG	1.1	42	0.64	140	4	2.27	5.1	2.86	273	115	109	7.7	NORMAL	NORMAL	NORMAL	SPUTUM C/S:ACINETOBACTER	NEG
185	140417	-	-	HMF ALTERED	NAD	NAD	NAD	10.6	6.5	87/9/5	127	66	NEG	1.53	40	0.9	130	3.5	0.6	6	3.2	78	890	675	8	MIXED ACID/BASE DISORDER	LEFT LZ HAZINESS	NORMAL	URINE C/S: KLEBSIELLA	NEG
186	982168	-	-	HMF ALTERED,RESPONSE TO PAIN ONLY	NAD	NAD	NAD	11.5	10.5	67/21/10	184	88	NA	1.34	49	1.03	132	4.69	0.22	5.4	2.33	73	35	42	7.8	NORMAL	NORMAL	NORMAL	1	NEG
187	986592	-	-	HMF ALTERED, B/L PLANTAR EXTENSOR	NAD	NAD	NAD	9.6	2.3	77/20/10/3	210	54	NEG	1.45	70	3.14	130	4	0.91	6	3.36	82	168	160	8.7	ACIDOSIS	INCREASED BRONCHOVASCULAR MARKINGS	NORMAL	URINE: E.COLI	NEG
188	1207450	-	-	HMF ALTERED	NAD	NAD	NAD	8.6	9.6	30/55/5	50	24	NEG	1.28	34	0.59	138	4	1.88	7.4	3.57	166	84	110	9.2	NORMAL	NORMAL	NORMAL	1	NEG
189	288933	-	-	HMF ALTERED	NAD	NAD	NAD	10.3	6.2	76/18/6	279	87	NEG	1	56	1.49	131	4.8	0.3	6.1	3.04	61	28	12	8.3	NORMAL	NORMAL	NORMAL	1	NEG
190	987800	-	-	INTUBATED,HMF ALTERED, MOVING ALL4 LIMBS, B/LPLANTAR FLEXOR	NAD	RIGHT ABSENT AIR ENTRY,LEFT SIDED CREPTS	NAD	7.9	16.4	65/20/10/4	561	54	NA	1.31	187	10.71	143	5.15	0.31	5.7	2.79	102	21	24	8.3	METABOLIC ACIDOSIS	RIGHT CAVITATORY LESION AND RIGHT PNEUMOTHORAX,PCD IN SITU	NORMAL	ET-ACINETOBACTER BAUMANII	NA
191	785234	-	-	HMF ALTERED	NAD	NAD	NAD	13	10	67/23/7/2	108	64	NEG	1.23	50	0.6	135	4	0.3	5.9	3	98	780	435	7.3	NORMAL	NORMAL	NORMAL	1	NEG
192	971448	-	-	HMF ALTERED, B/LPLANTAR EXTENSOR,MOVING ALL LIMBS	NAD	B/LDIFFUSE CREPTS	NAD	8.4	18.8	86/12/10	372	54	NA	1.19	24	0.6	139	4	0.68	4.8	2.77	154	32	18	8.9	ACIDOSIS,TYPE 1 RF	B/L SCATTERED INFILTRATES	NORMAL	1	NEG
193	990970	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	12.8	10.7	75/20/5	27	15	NEG	1.2	98	1.59	138	5	0.62	5.3	2.71	136	107	244	8.2	ACIDOSIS	NORMAL	NORMAL	1	NEG
194	990984	-	-	HMF ALTERED	NAD	B/L DIFFUSE SPASM	NAD	16	18.3	92/3/4	71	87	NEG	1.58	173	7.05	135	4	0.64	5.3	2.6	82	25	82	7.1	ACIDOSIS	B/L DIFFUSE INFILTRATES	NORMAL	1	NEG
195	991359	-	-	HMF ALTERED	NAD	B/L DIFFUSE SPASM	NAD	11.9	22	82/12/8	224	46	NEG	1.07	88	1.38	138	5.4	0.54	4.9	1.68	265	48	14	8.5	TYPE 1 RF	B/LDIFFUSEINFILTERATES	NORMAL	1	NEG
196	991446	-	-	HMF ALTERED	NAD	B/LBASAL AE REDUCED	NAD	8	14.2	86/8/6	80	12	NEG	1.11	113	2.3	142	4	0.92	6.6	3.7	175	30	29	8	ACIDOSIS	NORMAL	NORMAL	1	NEG
197	992248	-	-	PUPILS DILATED AND FIXED,DOLL'S EYE NEGATIVE,B/L PLANATAR MUTE	NAD	B/L AE REDUCED	NAD	7.8	10	86/22/2/2	128	24	NA	1.48	62	2.3	128	3.8	0.8	5.2	3	80	92	120	7.3	METABOLIC/RESPIRATORYACIDOSIS	ARDS	ABNORMAL	1	NEG
198	987795	-	-	HMF ALTERED	NAD	NAD	NAD	6.9	5.6	67/22/8/2	234	65	NEG	1.34	45	0.8	132	4	0.7	6.8	3.3	88	78	65	8.2	NORMAL	NORMAL	NORMAL	1	NEG
199	993161	-	-	HMF ALTERED	NAD	B/L REDUCED AIR ENTRY	NAD	9.8	13.8	85/9/6	146	73	NEG	1.09	94	4.77	139	4.62	0.37	5	2.26	73	17	15	8.3	ACIDOSIS	B/L PERIHILAR PROMINENCE	ABNORMAL	1	NEG
200	989223	Y	-	MENINGEAL SINGNS PRESENT,DROWSY BUT AROUSABLE	NAD	NAD	NAD	11.8	17.5	83/13/4	286	59	NEG	1.29	32	0.6	136	3.6	0.56	5.9	3.02	111	153	291	8	NORMAL	NORMAL	NORMAL	1	NEG
201	993199	-	-	HMF ALTERED	NAD	NAD	NAD	9	9.2	78/12/10	339	88	NEG	0.98	18	0.4	136	4.3	0.23	7.4	3.35	88	34	56	9	NORMAL	MILIARY MOTTLING	NORMAL	1	NA

S.NO	MRD NO	Leptospira	Dengue	Scrub Typhus	Other Investigations	CSF CYTOLOGY_TLC	NEUTROPHILS	LYMPHOCYTES	GLUCOSE	S. GLUCOSE	PROTEIN	CHLORIDE	GRAM STAIN	INDIA INK PREPARATION	BACTERIAL ANTIGEN	CULTURE SENSITIVITY	ADA	CBNAAT	HSV PCR	CT HEAD_FOCAL HYPODENSITIES	CT HEAD HYDROCEPHALUS	CT HEAD_DIFFUSE CEREBRAL OEDEMA	CT HEAD INFARCT	CT HEAD OTHERS	CT HEAD NORMAL	MRI LEPTOMENINGITIS	MRI VASCULITIC INFARCT
165	975285	NEG	NEG	NEG	-	2000	75	25	33	103	233	123	-	-	-	-	18.2	-	-	N	N	N	N	N	Y	Y	N
166	979358	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
167	977256	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
168	977162	NEG	NEG	NEG	PCT 64	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
169	980309	NEG	NEG	NEG	PCT35	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
170	978940	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
171	791852	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
172	975137	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
173	981400	NEG	POSITIVE	NEG	-	2	0	0	217	150	38	108	-	-	-	-	1.9	-	-	N	N	N	N	N	N	N	N
174	981849	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
175	980747	NEG	NEG	NEG	PCT 17,FUNGITELLPOSITIVE	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	Y
176	977161	NEG	POSITIVE	NEG	LVEF-52%	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
177	981093	NEG	POSITIVE	NEG	USG:POLYSEROSITIS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
178	982178	NEG	NEG	POSITIVE	PCT 5.9, URINE R/E - 80 PUS CELLS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
179	983103	NEG	NEG	NEG	PCT 0.48	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
180	983143	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
181	983854	NEG	NEG	NEG	-	2	0	0	76	99	56	108	-	-	-	-	4	-	NEGATIVE	N	N	N	N	N	N	N	N
182	980254	NEG	POSITIVE	NEG	PCT-21	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	Y	N	N	N	N	N
183	984893	NEG	NEG	NEG	PCT 60	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
184	213430	POSITIVE	NEG	NEG	PCT >100	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
185	140417	NEG	POSITIVE	NEG	USG:POLYSEROSITIS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
186	982168	EQUIVOCAL	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
187	986592	NEG	NEG	NEG	PCT >100	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	Y	CHRONIC INFARCT	N	N	N
188	1207450	NA	NA	POSITIVE	-	30	1	99	36	90	82	118	-	-	-	-	8.9	-	-	N	N	N	N	N	Y	N	N
189	288933	NA	NA	NA	PCT 8, URINE R/E: PUS CLESS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
190	987800	NA	NA	NA	PCT 89	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
191	785234	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	Y	N	N	N	N	N
192	971448	NEG	NEG	NEG	PCT-0.4,GALACTOMANNAN 0.48	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
193	990970	NEG	NEG	NEG	PCT 8.25	60	40	60	77	110	127	125	-	-	-	-	11.5	-	-	N	N	N	N	N	N	Y	N
194	990984	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
195	991359	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
196	991446	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	Y	N	N	N	N	N
197	992248	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	NA	N	N	N
198	987795	NA	NA	NA	-	5	0	100	46	78	127	117	-	-	-	-	12.7	POSITIVE	-	N	Y	N	N	DILATED TEMPORAL HORN	N	N	N
199	993161	NEG	NEG	NEG	PCT 28	2	0	0	115	120	35	109	-	-	-	-	2	-	-	N	N	N	N	N	Y	N	N
200	989223	NA	NA	NA	-	50	65	35	12	102	189	121	-	-	-	-	32	-	-	N	N	N	N	N	y	y	y
201	993199	NA	NA	NA	FUNDUS: CHROID TUBERCLES	20	10	90	58	110	62	108	-	-	-	-	10.2	-	-	N	N	N	N	N	N	N	N

S.NO	MRD NO	MRI_RING ENHANCING LESIONS	MRI_MENINGIOENCEPHALITIS/ ENCEPHALITIS	MRI HYDROCEPHALUS	MRI_DIFFUSE CERBRAL OEDEMA	MRI NORMAL	MRI_OTHERS	EEG (if needed)	TUBERCULOUS MENINGITIS	2.VIRAL	3.DENGUE ENCEPHALOPATHY/ ENCEPHALITIS	4.PYOGENIC	5.MALARIA	6.SCRUB RELATED ENCEPHALOPATHY	7.SEPTIC ENCEPHALOPATHY	POST COVID ENCEPHALOPATHY	LEPTOSPIRA INDUCED ENCEPHALOPATHY	OTHERS	TREATMENT GIVEN	OUTCOME 1. RECOVERED, 2. DAMA,
165	975285	N	Y	N	N	N	-	-	N	N	N	Y	N	N	N	N	N	N	IV ANTIBIOTICS,STEROIDS	1
166	979358	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,IV FLUIDS/SUPPORTIVE	1
167	977256	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV FLUIDS,ANTIBIOTICS,INOTROPES,VENTILATORY SUPPORT	3
168	977162	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
169	980309	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/INOTROPES/SUPPORTIVE	1
170	978940	N	N	N	N	N	ATROPHIC AND ISCHEMIC CHANGES	-	N	N	Y	N	N	N	N	N	N	N	IV FLUIDS/ANTIBIOTICS/STEROIDS/NAC	1
171	791852	N	N	N	N	Y	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS/INOTROPES/ MECHANICAL VENTILATORY SUPPORT	3
172	975137	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	STEROID/ANTIBIOTICS,IV FLUIDS,INOTROPES,VENTILATORY SUPPORT	3
173	981400	N	N	N	N	Y	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS/STEROIDS/SUPPORTIVE	1
174	981849	N	N	N	N	N	-	TRIPHASICWAVEPATTERN	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,INOTROPES,VENTILATORY SUPPORT	3
175	980747	N	N	N	N	N	CYTOTOXIC OEDEMA	GENERAL PERIODIC DISCHARGES	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ICD INSERTION/VENTILATORY SUPPORT	2
176	977161	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,STEROIDS,IV FLUIDS	1
177	981093	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV FLUIDS,VASOPRESSORS,OXYGEN SUPPORT	3
178	982178	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS,IV FLUIDS,IV INOTROPES	1
179	983103	N	N	N	N	Y	-	DIFFUSE ENCEPHALOPATHY	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
180	983143	N	Y	N	Y	N	TRANSIENT SPLENIALLESION	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRALS,FLUIDS/SUPPORTIVE	1
181	983854	N	N	N	N	N	CYSTIC ENCEPHALOMALACIA	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRALS/ANTIBIOTICS/CARDIAC MEDICINES	1
182	980254	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,FLUIDS,NAC,STEROIDS	3
183	984893	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS/SUPPORTIVE	3
184	213430	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS/FLUIDS	1
185	140417	N	N	N	N	Y	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,SIURETICS,STEROIDS,SUPPORTIVE TREATMENT	1
186	982168	N	N	N	Y	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV FLUIDS/STEROIDS/ANTI OEDEMA MEASURES	1
187	986592	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/INOTROPES/FLUIDS/SUPPORTIVE	2
188	1207450	N	Y	N	N	N	-	MODERATE DIFFUSE ENCEPHALOPATHY	N	N	N	N	N	Y	N	N	N	N	IV DOXY/RAMIPRIL/ANTIEPILEPTICS	1
189	288933	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
190	987800	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS,VENTILATORY SUPPORT, INOTROPES	1
191	785234	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV FLUIDS,ANTI OEDEMA/STEROIDS,SUPPORTIVE TREATMENT	1
192	971448	N	N	N	N	Y	-	-	N	N	N	N	N	N	N	Y	N	N	IV STEROIDS/IV ANTIBIOTICS/VENTILATOR/INOTROPES	3
193	990970	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/VENTILATOR	2
194	990984	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/VENTILATOR/DIURETICS	2
195	991359	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	2
196	991446	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIEPILEPTIC, IV ANTIBIOTIC, IV STEROIDS	1
197	992248	N	N	N	N	N	NA	-	N	Y	N	N	N	N	N	N	N	N	ATIBIOTICS/ANTIEPILEPTICS/ANTIVIRALS	3
198	987795	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROID	1
199	993161	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	2
200	989223	N	N	N	N	N	-	-	y	N	N	N	N	N	N	N	N	N	ATT/STEROID	1
201	993199	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1

S.NO	MRD NO	DOA	AGE	SEX	RELIGION	OCCUPATION	CHIEF COMPLAINTS FEVER	ALTERED SENSORIUM	HEADACHE	VERTIGO	BLURRING VISION	ABNORMAL BODY MOVEMENT	WEAKNESS IN ANY PART OF	VOMITING	OTHERS	PAST MEDICAL ILLNESS HISTORY			FAMILY HISTORY	PERSONAL HISTORY				SBP	DBP	TEMP	PULSE RATE	RR	PALOR	GCS	CLUBBING	ICTERUS	EDEMA	PETECHIAE / PURPURA
																TYPE 2 DM	HYPERTENSION	OTHERS		ALCOHOL	SMOKING	SUBSTANCE ABUSE	NONE										
202	993947	1-12-20	27	M	HINDU	AGRICULTURE	Y	Y	N	N	N	N	N	N	PAIN ABDOMEN	Y	-	-	NIL	-	-	-	Y	120	70	99.8	105	22	-	7	-	-	-	-
203	990192	1-12-20	38	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	70	40	101	129	18	-	5	-	-	-	-
204	992612	2-12-20	58	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	Y	N	-	-	-	-	NIL	Y	-	-	-	130	70	98	102	18	-	12	-	-	-	-
205	993575	4-12-20	55	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	Y	N	-	Y	-	-	NIL	-	-	-	Y	80	60	101	133	23	-	4	-	-	-	-
206	993401	5-12-20	65	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	80	50	102	122	18	Y	10	-	-	-	-
207	995076	6-12-20	52	M	HINDU	BUSINESS	Y	Y	N	N	N	N	Y	N	-	-	-	-	H/O POTT'S SPINE IN MOTHER	-	-	Y	-	100	60	98.6	102	22	-	12	-	-	-	-
208	208683	8-12-20	63	F	SIKH	TEACHER	Y	Y	N	N	N	N	N	N	SHORTNESS OF BREATH	Y	Y	-	DENGUE IN SON	-	-	-	Y	100	70	100	109	21	-	3	-	-	-	Y
209	998505	13/12/20	84	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	Y	-	-	-	-	H/O ABDOMINAL CANCER IN FATHER	-	-	-	Y	110	70	99	89	18	Y	11	-	-	-	-
210	997167	15/12/21	29	M	SIKH	SELF EMPLOYED	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	60	102	121	23	-	12	-	-	-	-
211	1001019	16/12/21	79	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	Y	-	NIL	-	-	-	Y	90	60	102	113	19	-	4	-	-	-	-
212	1002115	19/12/20	51	M	SIKH	BUSINESS	Y	Y	N	N	N	N	N	N	COUGH,HEMOPTYSIS	Y	-	-	PULMONARY KOCH'SIN SON	-	-	-	Y	110	70	101	118	27	Y	10	-	-	-	-
213	808357	19/12/20	55	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	JAUNDICE	-	-	CA GALL BLADDER	NIL	-	-	-	Y	110	70	101	134	18	Y	10	-	-	-	-
214	1002996	21/12/20	77	M	HINDU	LABOURER	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	80	50	99.8	114	18	-	10	-	-	-	-
215	998543	24/12/20	86	M	SIKH	BUSINESS	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	70	40	104	156	17	-	7	-	-	-	-
216	1005014	26/12/20	49	M	ISLAM	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	Y	-	70	40	102	156	30	Y	6	-	-	-	-
217	1001530	28/12/20	65	M	SIKH	LABOURER	Y	Y	N	N	N	N	N	N	LOW URINE OUTPUT	-	Y	-	NIL	-	-	-	Y	100	70	98.9	87	23	Y	6	-	-	-	-
218	993947	30/12/20	26	M	HINDU	STUDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	-	-	Y	110	70	102	98	18	Y	10	-	-	-	-
219	1001482	2-1-21	55	M	SIKH	SERVICE	Y	Y	Y	N	N	N	N	N	-	-	Y	CAD	NIL	-	-	-	Y	130	70	101	108	18	-	12	-	-	-	-
220	1007993	4-1-21	40	F	HINDU	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	120	70	101	107	18	-	12	-	-	-	-
221	1015877	21/1/21	45	F	HINDU	SELF EMPLOYED	Y	Y	Y	N	N	N	N	Y	-	-	-	HYPOTHYROID	NIL	-	-	-	Y	100	60	99	108	16	-	10	-	-	-	-
222	1014641	21/1/21	22	F	HINDU	STUDENT	Y	Y	Y	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	100	70	98.7	89	16	-	3	-	-	-	-
223	1016098	22/1/21	38	M	HINDU	BUSINESS	Y	Y	N	N	N	N	N	N	-	Y	Y	OLD KOCH'S	NIL	Y	-	-	-	90	60	96.3	87	18	Y	13	-	-	-	-
224	1018212	27/1/21	74	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	Y	-	-	-	90	50	100	145	16	-	12	-	-	-	-
225	1016823	28/1/21	85	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	Y	HYPOTHYROID	NIL	-	-	-	Y	90	60	98.6	98	18	-	11	-	Y	Y	-
226	995177	28/1/21	54	M	HINDU	BUSINESS	Y	Y	Y	N	Y	Y	N	N	-	Y	-	-	H/O PULMONARY TB IN SON	-	-	-	Y	80	50	99	120	16	-	11	-	-	-	-
227	837231	29/1/21	28	M	SIKH	SELF EMPLOYED	Y	Y	N	N	Y	Y	N	N	-	Y	-	-	NIL	Y	-	-	-	110	70	99.8	89	18	Y	10	-	-	-	-
228	999401	29/1/21	71	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	-	-	NIL	-	Y	-	-	100	70	99.8	102	22	-	12	-	-	-	-
229	1019436	30/1/21	78	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	DECREASED URINEOUTPUT	Y	-	CAD	NIL	-	-	-	Y	70	40	99	127	20	-	7	-	-	-	-
230	1024911	30/1/21	80	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	BURNING MICTURITION	Y	-	-	NIL	Y	-	-	-	140	79	100	98	16	-	11	-	-	-	-
231	1025438	10-2-21	28	M	SIKH	SELF EMPLOYED	Y	Y	N	N	N	N	Y	N	-	Y	-	-	NIL	-	-	-	Y	110	70	100	97	19	-	12	-	-	-	-
232	1070468	12-2-21	46	M	SIKH	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	100	60	100	99	21	-	11	-	-	-	-
233	1022005	12-2-21	35	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	PAIN ABDOMEN	-	-	-	NIL	-	-	-	Y	80	40	102	156	29	Y	8	-	Y	Y	-
234	922680	14/2/21	56	M	SIKH	SELF EMPLOYED	Y	Y	N	N	N	N	N	Y	-	-	-	LEFT SDH/BEDRIDDEN	NIL	-	Y	-	-	110	70	99.2	109	26	Y	9	-	-	Y	-
235	1028607	16/2/21	65	M	SIKH	SELF EMPLOYED	Y	Y	N	Y	N	N	N	N	-	Y	-	-	NIL	-	-	Y	-	80	30	102	166	34	-	5	-	-	Y	-
236	989223	19/2/21	46	F	SIKH	DEPENDENT	Y	Y	Y	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	100	60	100	97	20	Y	12	-	-	-	-
237	1026075	20/2/21	34	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	80	50	102	123	16	Y	3	-	-	-	-

S.NO	MRD NO	LYMPHADENOPATHY	CYANOSIS	CENTRAL NERVOUS SYSTEM	CVS	RS	P/A	HB	TLC	DLC	PLT	ESR	PBF MALARIA	PTI-INR	UREA	CREATININE	NA	K	S. BILIRUBIN	TOTAL PROTEIN	ALBUMIN	ALP	SGOT	SGPT	CALCIUM	ABG	CXR	ECG	BLOOD C/S/URINE C/S	Malaria Antigen
202	993947	-	-	HMF ALTERED,NO NECK RIGIDITY, NO FND/CND, B/LPLANTAR MUTE	NAD	NAD	NAD	11.9	8.3	92/6/2	379	28	NEG	0.98	30	0.61	141	4.6	0.05	6.1	3.23	135	95	150	8.7	METABOLIC ACIDOSIS	ENHANCED BROCHOVASCULAR MARKINGS	NORMAL	1	NEG
203	990192	-	-	HMF ALTERED/MENINGEAL SINGS PRESENT,B/L PLNATAR MUTE	NAD	NAD	HEPATOMEGALY,ABDOMEN DISTENDED	8.4	13.9	70/20/7/2	24	77	NEG	1.34	55	1.38	146	4.4	1.79	5.3	1.78	371	117	37	7.8	SEVERE METABOLIC AND LACTIC ACIDOSIS	B/L ARDS	ABNORMAL	PUS C/S: E.COLI	NEG
204	992612	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	11.8	8.3	65/22/10	266	26	NEG	1.23	19	0.97	132	5.7	0.4	7	3.6	88	22	16	8.2	NORMAL	NORMAL	NORMAL	1	NEG
205	993575	-	-	HMF ALTERED,INTUBATED,LEFT HEMIPARESIS	NAD	AE REDUCED IN RIGHT SIDE	NAD	10.2	11.9	85/9/6	347	87	NEG	1.26	96	0.57	135	3.8	0.4	5.5	2.45	80	57	82	8.3	METABOLICACIDOSIS	RIGHT LZ HAZINESS	ABNORMAL	1	NEG
206	993401	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	10	10.3	70/18/10	259	12	NEG	1.21	29	0.42	143	3.5	0.92	7.7	3.17	71	31	30	9	ACIDOSIS	NORMAL	NORMAL	1	NEG
207	995076	Y	-	HMF ALTERED,RIGHT HEMIPARESIS	NAD	NAD	NAD	9.5	8.2	68/22/8/2	319	82	NEG	1.45	30	0.58	133	3.6	0.46	6.5	3.76	49	19	18	8	NORMAL	NORMAL	NORMAL	1	NEG
208	208683	-	-	HMF ALTERED,INTUBATED, B/L PUPILS FIXED	NAD	B/L LZ AIR ENTRY DECREASED	NAD	8.3	21.3	67/22/8	137	45	NEG	1.43	60	0.83	143	3.99	0.52	6.1	2.89	229	637	541	6.9	ACIDOSIS	B/L DIFFUSE FLUFFY SHADOWS	ABNORMAL	ET-ACINETOBACTER	NEG
209	998505	-	-	HMF ALTERED	NAD	NAD	NAD	8.2	19.1	88/8/4	118	88	NEG	1.19	73	1.54	138	3.8	11.3	4.8	3.05	555	87	55	8.2	NORMAL	NORMAL	NORMAL	URINE C/S: E.COLI	NEG
210	997167	-	-	HMF ALTERED	NAD	NAD	NAD	12	10	68/22/10	152	18	NA	1.02	19	0.8	134	3.8	0.06	6.7	4.03	56	37	14	8.1	NORMAL	NORMAL	NORMAL	1	NEG
211	1001019	-	-	HMF ALTERED,DECEREBRATING,B.L PLANTAR MUTE	NAD	B/L SCATTERED CREPTS	NAD	12.1	9.4	88/12	194	32	NA	1.03	55	1.79	133	7	1.37	6.6	3.15	142	1896	1784	8.1	LACTIC ACIDOSIS	PERI HILAR FLUFFY SHADOWS	ABNORMAL	1	NEG
212	1002115	-	-	HMF ALTERED	NAD	BRONCHIAL BREATHINGONLEFT SIDE	NAD	14.2	22.2	96/3/1	348	120	NEG	1.33	87	2.27	143	4.3	0.17	5	2.07	79	36	16	8.9	ALKALOSIS	LEFT LOBAR PNEUMONIA WITH CAVITY	NORMAL	1	NEG
213	808357	-	-	HMF ALTERED	NAD	NAD	TENDER	6.8	2.3	67/28/5	10	110	NEG	1.02	68	1.53	131	4	4.16	4.5	1.92	713	1242	637	8	NORMAL	NORMAL	NORMAL	PUS C/S: E.COLI	NEG
214	1002996	-	-	HMF ALTERED,B/L PLANTAR UPGOING	NAD	NAD	NAD	7.9	24.2	80/16/4	276	100	NEG	1.09	115	6.4	138	3.8	0.56	5.7	2.49	156	45	89	7.9	ACIDOSIS	B/L PLEURAL EFFUSION	NORMAL	URINE C/S: ENTEROCOCUS FAECIUM	NEG
215	998543	-	-	HMF ALTERED, B/LPLANTAR MUTE	NAD	NNAD	TENSE,TENDER,GUARDING,RIGIDITY	12.7	14.5	80/10/8	295	54	NA	1	72	1.83	145	4.2	0.43	6.7	3.51	85	51	31	8.9	SEVERE MIXED ACIDOSIS	NORMAL	ABNORMAL	PCD FLUID:KLEBSIELLA	NEG
216	1005014	-	-	HMF ALTERED	NAD	B/L CONDUCTED SOUNDS	NAD	6.7	20	90/8/2	110	53	NEG	1.37	241	10	132	8	2.55	5.2	2.6	130	47	92	6.5	ACIDOSIS	B/L DIFFUSE INFILTRATES	ABNORMAL	BLOOD C/S: MRSA	NEG
217	1001530	-	-	HMF ALTERED	NAD	B/L SCATTERED CREPTS	NAD	9.3	10.3	88/6/5	80	62	NEG	1.6	265	8.78	146	5.3	0.9	5.9	3.01	125	3587	1991	7.9	METABOLIC ACIDOSIS	B/L LZ HAZINESS/PERIHILAR OPACITIES	NORMAL	1	NEG
218	993947	-	-	HMF ALTERED	NAD	NAD	NAD	12.5	4.5	61/26/8	290	98	NEG	1.19	19	0.68	137	4.48	0.33	6.5	3.47	121	22	44	8.9	ACIDOSIS	NORMAL	NORMAL	1	NEG
219	1001482	-	-	HMF ALTERED	ABNORMAL	NAD	NAD	11	6.7	90/5/5	110	65	NEG	1.3	30	0.9	138	4.9	1.96	5.6	2.84	268	84	56	6.8	NORMAL	B/L EFFUSION	LBBB	1	NEG
220	1007993	-	-	HMF ALTERED	NAD	NAD	NAD	10.9	9.2	73/20/7	148	16	NA	1.02	15	0.55	141	4	0.31	6.9	4.07	62	22	18	8.2	NORMAL	NORMAL	NORMAL	1	NEG
221	1015877	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	11.8	12.3	97/1.5/2	214	67	NA	1.3	62	0.78	136	0.9	0.37	6	3.5	123	40	50	9.2	NORMAL	NORMAL	NORMAL	1	NA
222	1014641	-	-	HMF ALTERED, NO RESPONSE TO PAIN, B/L PLANTAR MUTE, DOLL'SEYE REFLEX ABSENT	NAD	NAD	NAD	10.8	11.2	73/17/7	344	17	NEG	0.95	23	0.44	140	3.2	0.46	4.9	2.7	486	24	24	8.4	MIXED ACIDOSIS	ABNORMAL	ABNORMAL	1	NEG
223	1016098	-	-	HMF ALTERED,MENINGEALSIGNS	NAD	NAD	NAD	8.2	5.8	67/21/2	189	15	NEG	1.24	14	0.23	135	0.4	0.27	7.4	4.5	75	45	34	8	NORMAL	NORMAL	NORMAL	1	NEG
224	1018212	Y	-	HMF ALTERED	NAD	LEFT AIR ENTRY REDUCED	NAD	7.7	5.4	73/16/6	70	18	NEG	1.21	119	3.87	143	5	2.25	5.3	1.74	232	428	91	8.2	ACIDOSIS	LEFT PLEURAL EFFUSION	NORMAL	BLOOD C/S:MRSA	NEG
225	1016823	-	-	HMF ALTERED,NECK RIGIDUTYPRESENT	ABNORMAL	NAD	NAD	11.3	48.3	95/1/3	68	54	NEG	1.08	124	2.14	141	3.8	4.44	5.3	2.4	964	124	131	9	ACIDOSIS	NORMAL	NORMAL	URINE C/S: E. COLI	NEG
226	995177	-	-	MENINGEAL SIGNS,ALTERED HMF	NAD	NAD	NAD	12.2	8.6	69/22/8	162	80	NEG	1.26	26	0.3	132	3.8	0.38	6.5	3.6	82	181	78	8.2	NORMAL	NORMAL	NORMAL	1	NA
227	837231	Y	-	HMF ALTERED ,LEFT PLANTAR UPGOING	NAD	NAD	NAD	8.9	14	76/17/3	190	78	NA	1.39	78	1.9	133	4	0.67	6.7	3.2	88	57	42	7.6	METABOLIC ACIDOSIS	B/L LOWER ZONEHAZINESS	NORMAL	1	NA
228	999401	-	-	HF ALTERED	NAD	B/L AIR ENTRY REDUCED	NAD	10.9	21	92/6/2	145	21	NEG	1.09	167	4.14	132	5	0.7	7	3.1	156	34	890	6.9	ACIDOSIS	B/L LZ HAZINESS	NORMAL	1	NEG
229	1019436	-	-	HMFALTERED, B/L PLANTAR MUTE	ABNORMAL	B/L CREPTS PRESENT	NAD	10	15.4	95/4/1	110	34	NEG	1.23	126	3.35	146	5	1.43	4.4	2.07	200	36	31	8.5	ACIDOSIS	DIFFUSE FLUFFY SHADOWS	ABNORMAL	1	NEG
230	1024911	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	13.4	6.9	92/4/3	124	86	NA	1.34	73	0.65	129	4.31	0.6	7.2	4.3	78	103	78	8	NORMAL	NORMAL	NORMAL	1	NA
231	1025438	-	-	DROWSY,AROUSABLE, PARAPARESIS	NAD	NAD	NAD	13.1	10.5	90/7/2/1	160	54	NEG	1.29	26	0.98	137	4	0.39	6.9	4	83	33	37	9.2	NORMAL	NORMAL	NORMAL	1	NEG
232	1070468	-	-	HMF ALTERED	NAD	NAD	NAD	9.4	6.4	90/7/2/1	22	28	NEG	1.73	123	3.72	134	5.3	1.09	7	3.41	87	36	48	8.5	ACIDOSIS	PERI HILAR OPACITIES	NORMAL	URINE C/S: E.COLI	NEG
233	1022005	-	-	HMF ALTERED, B/LPLANTARMUTE	ABNORMAL	B/LDIFFUSE CREPTS	NAD	7	16.4	95/2.4	150	89	NEG	1.45	67	1.61	139	4.6	0.67	5.5	2.3	139	34	27	9.2	ACIDOSIS,TYPE 1 RF	B/L DIFFUSE INFILTRATES	ABNORMAL	1	NEG
234	922680	-	-	HMF ALTERED, B/LPLANTAR EXTENSOR	NAD	RIGHT SIDED COARSE CREPTS	NAD	11.1	9.1	68/22/6/4	350	22	NA	1.28	18	0.41	132	40	0.47	7.1	3.36	284	20	73	8	TYPE1RF	B/LLOWERZONES HAZINESS	NORMAL	1	NA
235	1028607	-	-	HMF ALTERED, B/L PLANTAR MUTE,DECREBRATING	NAD	B/LCREPTS HEARD	NAD	13.5	30	85/7/8	356	12	NEG	1.17	117	3.52	145	4	0.54	6.7	3	135	21	21	8.3	ACIDOSIS	B/LLOWER ZONE HAZINESS	ABNORMAL	1	NEG
236	989223	Y	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	B/L FINE CREPTS	NAD	7.4	9.2	70/22/8	162	88	NEG	1.32	27	1.1	133	3.8	0.42	4.9	2.26	82	228	321	8.2	NORMAL	NORMAL	NORMAL	1	NA
237	1026075	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT,B/LPLANTAR EXTENSOR	NAD	NAD	NAD	7.8	13.2	82/17/1	167	56	NEG	1.34	33	0.6	137	4.2	0.54	7.2	3.6	444	192	282	10.1	NORMAL	NORMAL	NORMAL	1	NA

S.NO	MRD NO	Leptospira	Dengue	Scrub Typhus	Other Investigations	CSF CYTOLOGY_TLC	NEUTROPHILS	LYMPHOCYTES	GLUCOSE	S. GLUCOSE	PROTEIN	CHLORIDE	GRAM STAIN	INDIA INK PREPARATION	BACTERIAL ANTIGEN	CULTURE SENSITIVITY	ADA	CBNAAT	HSV PCR	CT HEAD_FOCAL HYPODENSITIES	CT HEAD HYDROCEPHALUS	CT HEAD_DIFFUSE CEREBRAL OEDEMA	CT HEAD INFARCT	CT HEAD OTHERS	CT HEAD NORMAL	MRI LEPTOMENINGITIS	MRI VASCULITIC INFARCT
202	993947	NA	NA	NA	PCT - 0.2	2	0	0	87	102	26	124	-	-	-	-	1.5	-	NEGATIVE	N	N	N	N	N	N	N	N
203	990192	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
204	992612	NA	NA	NA	-	400	10	90	59	107	257	112	-	-	-	-	26	-	-	N	N	N	N	N	N	N	N
205	993575	NEG	NEG	NEG	-	200	20	80	28	87	127	108	-	-	-	-	86	-	-	N	N	N	N	N	N	Y	Y
206	993401	NEG	NEG	NEG	-	200	70	30	2	90	1663	110	-	-	-	SALMONELLATYPHI	-	NEGATIVE	-	N	N	N	N	N	N	Y	N
207	995076	NEG	NEG	NEG	-	90	10	90	40	138	160	113	-	-	-	-	-	POSITIVE	-	N	N	N	N	N	N	Y	Y
208	208683	NEG	POSITIVE	NEG	-	10	30	70	68	145	34.5	134	-	-	-	-	5.44	-	-	N	N	N	N	N	N	N	N
209	998505	NEG	NEG	NEG	TUMOR MARKERS:HIGH, USG-PERIPANCREATIC MASS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
210	997167	NEG	NEG	NEG	-	10	0	100	52	98	47	120	-	-	-	-	2	-	POSITIVE	N	N	N	N	N	N	N	N
211	1001019	NEG	NEG	NEG	-	2	0	0	84	89	78	114	-	-	-	-	2.72	-	NEGATIVE	N	N	N	N	N	N	N	N
212	1002115	NEG	NEG	NEG	PCT-0.20	2	0	0	154	280	47	108	-	-	-	-	2.3	-	-	N	N	N	N	N	N	N	N
213	808357	NEG	NEG	NEG	PCT 38, CHOLANGITIS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
214	1002996	NA	NA	NA	PCT 2.4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	Y
215	998543	NA	NA	NA	PCT >100, EMPHYSEMATOUS CHOLECYSTITIS ?	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
216	1005014	NEG	NEG	NEG	PCT 68	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
217	1001530	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
218	993947	NEG	NEG	NEG	PCT-0.19, COVID ANTIBODY POSITIVE	5	0	0	87	157	25	124	-	-	-	-	1.74	-	NEGATIVE	N	N	N	N	N	Y	N	N
219	1001482	NEG	POSITIVE	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
220	1007993	NEG	NEG	NEG	-	2	0	0	85	89	75	125	-	-	-	-	2.14	-	NEGATIVE	N	N	N	N	N	N	N	N
221	1015877	NA	NA	NA	-	300	20	80	18	86	162	118	-	-	-	-	189	-	-	N	N	N	N	N	N	N	N
222	1014641	NEG	NEG	NEG	-	50	30	70	63	90	56	121	-	-	-	-	23.2	-	NEGATIVE	N	Y	Y	N	N	N	N	N
223	1016098	NA	NA	NA	-	10	5	95	88	123	84	112	-	-	-	-	4	-	NEGATIVE	N	N	N	N	N	N	N	N
224	1018212	NEG	NEG	NEG	PCT-11.18	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
225	1016823	NEG	NEG	NEG	USG- DILATED CBD AND MPD WITH BILOBAR IHBRD	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
226	995177	NA	NA	NA	-	80	20	80	18	182	92	112	-	-	-	-	18	POSITIVE	-	N	N	N	N	N	N	N	N
227	837231	NA	NA	NA	-	90	10	90	45	96	154	108	-	-	-	-	27	-	-	N	Y	N	N	N	N	N	N
228	999401	NA	NA	NA	PCT15	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
229	1019436	NEG	NEG	NEG	PCT-6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
230	1024911	NA	NA	NA	-	2	0	0	28	102	154	109	-	-	-	-	26	-	-	N	Y	N	N	N	N	Y	Y
231	1025438	NEG	NEG	NEG	ANTI KU ANTIBODY POSITIVE	-	-	-	-	-	-	-	-	POSITIVE	-	-	-	-	-	N	N	N	N	N	N	N	N
232	1070468	NA	NA	NA	FUNGITELL POSITIVE	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
233	1022005	POSITIVE	NEG	NEG	PCT-4.1, SEVER LV DYSFUNCTION	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
234	922680	NA	NA	NA	CT: B/L CONSOLIDATION ?	2	0	0	83	102	114	121	-	-	-	-	1.3	-	-	N	N	N	N	ENCEPHALOMALACIA/POST OP CHANGES	N	N	N
235	1028607	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
236	989223	NA	NA	NA	-	300	10	90	36	106	88	102	-	-	-	-	28	-	-	N	N	N	N	N	N	N	Y
237	1026075	NA	NA	NA	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	Y	Y	N	N	N	N	N	N

S.NO	MRD NO	MRI_RING ENHANCING LESIONS	MRI_MENINGIOENCEPHALITIS/ ENCEPHALITIS	MRI HYDROCEPHALUS	MRI_DIFFUSE CERBRAL OEDEMA	MRI NORMAL	MRI_OTHERS	EEG (if needed)	TUBERCULOUS MENINGITIS	2.VIRAL	3.DENGUE ENCEPHALOPATHY/ ENCEPHALITIS	4.PYOGENIC	5.MALARIA	6.SCRUB RELATED ENCEPHALOPATHY	7.SEPTIC ENCEPHALOPATHY	POST COVID ENCEPHALOPATHY	LEPTOSPIRA INDUCED ENCEPHALOPATHY	OTHERS	TREATMENT GIVEN	OUTCOME 1. RECOVERED, 2. DAMA,
202	993947	N	N	N	N	Y	-	MODERATE DIFFUSE ENCEPHALOPATHY	N	N	N	N	N	N	N	N	N	N	STEROIDS/ANTIEPILEPTICS/ANTIBIOTICS	1
203	990192	N	N	N	N	Y	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,IV STEROIDS,VENTILATORY SUPPORT	2
204	992612	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROID	1
205	993575	N	Y	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STROIDS	1
206	993401	N	N	N	N	N	VENTRICULITIS	-	N	N	N	Y	N	N	N	N	N	S. TYPHI MENINGITIS	IV ANTIBIOTICS/VENTILATOR/DIURETICS	3
207	995076	Y	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
208	208683	N	N	N	N	Y	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,INOTROPES,IV FLUIDS,VENTILATORY SUPPORT	1
209	998505	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS,UDIILIV,NAC	3
210	997167	N	Y	N	N	N	S/O HERPES ENCEPHALITIS	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRAL,ANTIEPILEPTIC	1
211	1001019	N	N	N	N	N	RIGHT FRONTAL LOBEGYRIFORM DIFFUSION RESTRICTION	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIBIOTICS/ANTI VIRAL/INOTROPES/MECHANICAL VENTILATORY SUPPORT	3
212	1002115	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	ATT/IV ANTIBIOTICS	1
213	808357	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/FLUIDS	3
214	1002996	N	N	N	N	N	?SEPTIC EMBOLI	-	N	N	N	N	N	N	Y	N	N	N	IV ANTINIOTICS/INOTROPES/FLUIDS	2
215	998543	N	N	N	N	N	-	DIFFUSE ENCEPHALOPATHY	N	N	N	N	N	N	Y	N	N	N	IVANTIBIOTICS, ABDOMINALPCD INSERTION, INOTROPES,VENTILATORY SUPPORT	3
216	1005014	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS,VENTILATORY SUPPORT, INOTROPES	3
217	1001530	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	IV ANTIBIOTICS,FLUIDS,STEROIDS,TRENA	1
218	993947	N	N	N	N	N	B/L HIPPOCAMPI WITH DIFFUSION RESTRICTION, ?POST ICTAL	-	N	N	N	N	N	N	N	Y	N	N	IV ANTIEPILEPTIC, IV ANTIBIOTIC, IV STEROIDS	2
219	1001482	N	N	N	N	N	-	-	N	N	Y	N	N	N	N	N	N	N	STEROIDS,IV FLUIDS	1
220	1007993	N	N	N	N	Y	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRALS/	3
221	1015877	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/ANTIEPILEPTICS/ANTIBIOTICS	1
222	1014641	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROID/IV ANTIBIOTICS/ANTI EODEMA	3
223	1016098	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRAL/IV ANTIBIOTICS	3
224	1018212	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/UDILIV/IV FLUIDS	3
225	1016823	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/INOTROPES/CNS STIMULANTS	3
226	995177	N	Y	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	STEROIDS/ENDOXAN PULSE/ATT	1
227	837231	Y	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/ANTI OEDEMA MEASURES	2
228	999401	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
229	1019436	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS,CARDIAC MEDICATION,PERITONEALDIALYSIS	3
230	1024911	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/MECHANICAL INTUBATION	3
231	1025438	N	N	N	N	N	-	-	N	N	N	N	N	N	N	N	N	cryptococcal meningitis	ANTIEPILEPTIC/STROID	1
232	1070468	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIFUNGAL/ ANTIBIOTICS,INOTROPES,SEPTIC DOSE STEROIDS	1
233	1022005	N	N	N	N	N	-	MODERATE DIFFUSE ENCEPHALOPATHY	N	N	N	Y	N	N	N	N	N	N	IV ANTIBIOTICS/LAPROTOMY	3
234	922680	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ANTIFUNGALS/SUPPORTIVE	3
235	1028607	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/PERITONEAL DIALYSIS/VNTILATORY SUPPORT	3
236	989223	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	MODIFIED ATT/STEROIDS	1
237	1026075	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	MODIFIED ATT/STEROIDS	3

S.NO	MRD NO	DOA	AGE	SEX	RELIGION	OCCUPATION	CHIEF COMPLAINTS FEVER	ALTERED SENSORIUM	HEADACHE	VERTIGO	BLURRING VISION	ABNORMAL BODY MOVEMENT	WEAKNESS IN ANY PART OF	VOMITING	OTHERS	PAST MEDICAL ILLNESS HISTORY			FAMILY HISTORY	PERSONAL HISTORY				SBP	DBP	TEMP	PULSE RATE	RR	PALOR	GCS	CLUBBING	ICTERUS	EDEMA	PETECHIAE / PURPURA
																TYPE 2 DM	HYPERTENSION	OTHERS		ALCOHOL	SMOKING	SUBSTANCE ABUSE	NONE											
238	1034107	24/2/21	85	F	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	Y	Y	OLD KOCH'S	NIL	-	-	-	Y	110	70	100	94	20	-	12	-	-	-	-
239	1042343	5-3-21	68	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	99	110	18	-	12	-	-	-	-
240	1043963	7-3-21	70	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	PAIN ABDOMEN	-	Y	CAD	NIL	Y	-	-	-	120	70	99	100	18	-	12	-	-	-	-
241	1045405	9-3-21	55	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	Y	DECREASED URINE OUTPUT,PAIN ABDOMEN	Y	-	-	NIL	-	-	-	Y	70	40	100	165	28	-	10	-	-	-	-
242	1050401	15/3/21	56	M	SIKH	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	LEFT LEG CELLULITIS	Y	-	-	NIL	Y	-	-	-	60	40	101	145	33	-	7	-	-	Y	-
243	1059160	20/3/21	38	F	SIKH	DEPENDENT	Y	Y	Y	N	N	N	N	Y	-	-	-	-	PULMONARY KOCH'S ON BROTHER	-	-	-	Y	100	60	101	117	15	Y	11	-	-	-	-
244	1022929	20/3/21	59	M	SIKH	RETIRED	Y	Y	N	N	N	N	N	N	PYURIA	Y	-	-	NIL	-	-	-	Y	90	60	100	89	18	-	12	-	-	Y	-
245	1065734	26/3/21	58	M	SIKH	BUSINESS	Y	Y	N	N	N	N	N	N	-	-	Y	-	NIL	-	-	-	Y	150	70	99.8	92	16	-	12	-	-	-	-
246	891968	31/3/21	43	M	HINDU	BUSINESS	Y	Y	N	N	N	Y	N	N	-	-	-	ALL	NIL	-	-	-	Y	100	60	101	134	22	-	9	-	-	-	-
247	1020516	31/3/21	18	M	SIKH	STUDENT	Y	Y	Y	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	120	70	100	86	18	-	12	-	-	-	-
248	1072015	1-4-21	60	M	SIKH	RETIRED	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	100	60	101	99	18	-	12	-	-	-	-
249	993575	2-4-21	55	M	SIKH	AGRICULTURE	Y	Y	N	N	N	N	N	N	-	Y	Y	-	NIL	-	-	-	Y	110	70	98	78	18	-	12	-	-	-	-
250	1079544	8-4-21	61	M	SIKH	BUSINESS	Y	Y	N	N	N	N	N	N	CONSTIPATION	-	-	-	NIL	-	-	-	Y	120	60	100	85	17	-	12	-	-	-	-
251	1084302	12-4-21	21	M	HINDU	AGRICULTURE	Y	Y	N	N	N	N	N	N	B/L LOWER LIMB WEAKNESS	Y	-	-	NIL	-	-	-	Y	100	70	101	123	21	-	12	-	-	-	-
252	1094189	24/4/21	24	M	SIKH	STUDENT	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	103	122	19	-	12	-	-	-	-
253	1081599	30/4/21	26	M	SIKH	STUDENT	Y	Y	N	N	N	N	N	N	-	Y	Y	-	NIL	-	-	-	Y	100	70	100	121	20	-	12	-	-	-	-
254	1114293	28/5/21	40	M	SIKH	BUSINESS	Y	Y	N	N	N	Y	N	N	-	Y	-	-	NIL	-	-	-	Y	100	60	101	132	22	-	11	-	-	-	-
255	1107438	28/5/21	55	M	SIKH	AGRICULTURE	Y	Y	N	N	Y	N	Y	N	-	-	-	-	NIL	-	-	-	Y	130	70	101	102	18	-	12	-	-	-	-
256	1138940	28/5/21	45	M	SIKH	SELF EMPLOYED	Y	Y	Y	N	N	N	N	Y	-	-	-	OLD KOCH'S	NIL	-	-	Y	-	110	70	100	118	19	-	10	-	-	-	-
257	1137033	30/5/21	33	M	HINDU	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	120	80	99.4	98	18	-	8	-	-	-	-
258	1137166	31-05-2021	52	M	HINDU	SELF EMPLOYED	Y	Y	Y	N	N	N	N	N	-	N	N	COAD	NIL	Y	-	-	-	110	70	100	98	16	-	11	-	-	-	-
259	965022	1-6-21	41	M	MUSLIM	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	DECREASED URINEOUTPUT	-	-	ADPKD	NIL	-	-	-	Y	100	70	101	117	18	-	12	-	-	-	-
260	726920	4-6-21	87	M	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	ICTERUS	-	Y	-	NIL	Y	-	-	-	130	60	99.9	89	20	Y	10	-	-	-	-
261	1119623	8-6-21	32	M	HINDU	SELF EMPLOYED	Y	Y	N	N	N	N	N	N	SHORTNESS OF BREATH	-	-	OSA	NIL	Y	Y	-	-	110	60	98	88	21	-	6	-	-	-	-
262	1111684	8-6-20	52	M	SIKH	BUSINESS	Y	Y	N	N	N	Y	N	N	-	Y	Y	POST COVID	NIL	Y	-	-	-	100	70	100	99	18	-	9	-	-	-	-
263	1120428	9-6-21	87	F	SIKH	DEPENDENT	Y	Y	N	N	N	N	N	N	INCREASED FREQUENCY OF MICTURITION	-	Y	-	NIL	-	-	-	Y	130	70	99	102	20	Y	11	-	-	-	-
264	1120986	10-6-21	53	M	MUSLIM	LABOURER	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	110	70	97.8	76	16	-	11	-	-	-	-
265	1119436	11-6-21	22	M	SIKH	SELF EMPLOYED	Y	Y	N	N	N	Y	N	N	-	-	-	HIV	NIL	-	-	Y	-	90	60	102	134	22	-	10	-	-	-	-
266	1060293	21/6/21	65	M	HINDU	DEPENDENT	Y	Y	N	N	N	N	N	N	PYURIA	Y	-	CAD	NIL	-	-	-	Y	90	60	99.6	89	18	-	10	-	-	Y	-
267	1133070	25/6/21	45	M	SIKH	SERVICE	Y	Y	N	N	N	N	N	N	-	-	-	-	NIL	-	-	-	Y	90	50	100	89	17	-	12	-	-	-	-
268	1128159	26/6/21	33	M	HINDU	SERVICE	Y	Y	N	N	N	Y	N	Y	-	-	-	-	NIL	-	-	-	Y	100	60	98.3	108	16	-	12	-	-	-	-
269	955938	30/6/21	25	F	SIKH	SELF EMPLOYED	Y	Y	N	N	N	N	N	Y	-	-	-	-	NIL	-	-	-	Y	90	50	100	108	16	Y	12	-	-	-	-
270	1133584	30/6/21	72	M	HINDU	DEPENDENT	Y	Y	N	N	N	Y	N	Y	-	Y	-	-	NIL	-	-	-	Y	80	60	99.2	99	32	-	8	-	-	-	-
271	1126690	24-06-2021	27	F	SIKH	DEPENDENT	Y	Y	N	N	N	Y	N	N	-	-	-	-	NIL	-	-	-	Y	100	60	104	134	18	-	12	-	-	-	-

S.NO	MRD NO	LYMPHADENOPATHY	CYANOSIS	CENTRAL NERVOUS SYSTEM	CVS	RS	P/A	HB	TLC	DLC	PLT	ESR	PBF MALARIA	PTI-INR	UREA	CREATININE	NA	K	S. BILIRUBIN	TOTAL PROTEIN	ALBUMIN	ALP	SGOT	SGPT	CALCIUM	ABG	CXR	ECG	BLOOD C/S/URINE C/S	Malaria Antigen
238	1034107	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	LEFT AE REDUCED	NAD	12.8	8	82/10	253	120	NEG	1.19	38	0.73	131	4.8	0.38	5.5	2.52	81	93	45	9.8	NORMAL	LEFT PLEURAL EFFUSION	NORMAL	1	NA
239	1042343	-	-	HMF ALTERED,MENINGEALSIGNS PRESENT	NAD	NAD	NAD	12.4	29.2	93/6/1	140	11	NEG	1.56	24	0.74	134	3.7	0.79	7.1	3.61	107	22	16	9	NORMAL	NORMAL	NORMAL	1	NEG
240	1043963	-	-	HMF ALTERED,NUCHAL RIGIDITY	NAD	NAD	NAD	14.8	8.2	72/15/10	198	12	NEG	1.19	41	0.9	141	4	0.39	5.6	3.23	62	36	52	9.2	NORMAL	NORMAL	NORMAL	1	NEG
241	1045405	-	-	HMF ALTERED,RESTLESS, B/L PLANTAR WITHDRAWL	NAD	B/L AIR ENTRY REDUCED	NAD	12.1	16.2	88/6/4/2	60	49	NEG	1.13	148	5.41	137	6	2.67	5.3	2.56	160	88	47	9	ACIDOSIS	B/LPLEURAL EFFUSION,RIGHT LOWERZONE PATCH	ABNORMAL	1	NEG
242	1050401	-	-	HMF ALTERED	NAD	NAD	NAD	11.5	6.8	85/9/5	123	24	NEG	1.23	126	5.8	148	5.8	1.13	6.2	3.5	278	78	90	8.1	ACIDOSIS	B/LPLEURAL EFFUSION	ABNORMAL	WOUND C/S: ENTEROCOCCUS	NEG
243	1059160	-	-	HMF ALTERED,NUCHAL RIGIDITY	NAD	NAD	NAD	7.6	11	67/20/3	189	18	NEG	1.07	34	0.5	136	3.8	0.35	7	3.6	78	45	24	8.4	NORMAL	NORMAL	NORMAL	1	NA
244	1022929	-	-	HMF ALTERED	NAD	NAD	NAD	12	4	88/6/5	159	22	NEG	1.18	31	0.62	133	3.7	0.71	5.3	3.13	78	18	18	9	NORMAL	NORMAL	NORMAL	URINE C/S:PSEUDOMONAS	NA
245	1065734	-	-	HMF ALTERED	NAD	NAD	NAD	15.2	7.8	78/11/10	158	46	NEG	1.26	56	0.98	145	3.8	1.96	7.5	4.81	129	262	272	9	NORMAL	NORMAL	NORMAL	1	NEG
246	891968	-	-	HMF ALTERED, LEFT HEMIPARESIS	NAD	NAD	NAD	6.6	2.3	70/23/6	40	102	NEG	1.2	65	0.84	135	3.8	0.8	6.1	2.77	89	23	45	9	ALKALOSIS	B/LDIFFUSEINFILTERATES	NORMAL	1	NEG
247	1020516	-	-	HMF ALTERED	NAD	NAD	NAD	12.3	8	78/12/8	245	67	NEG	1.25	22	0.5	134	3.8	0.21	6.8	3.8	58	23	18	7.9	MILLIARY SHADOWS	NORMAL	NORMAL	1	NA
248	1072015	-	-	HMF ALTERED	NAD	NAD	NAD	14.8	13.7	83/9/7	190	43	NEG	1.29	23	0.78	135	4.05	0.38	5.7	3.08	51	86	116	7.9	ACIDOSIS	NORMAL	NORMAL	1	NEG
249	993575	-	-	HMF ALTERED	NAD	NAD	NAD	11.4	6.2	72/16/2	185	23	NEG	1.03	23	0.4	135	4	0.45	6.9	4.1	87	23	34	8.9	NORMAL	NORMAL	NORMAL	URINE R/E 25PUS CELLS	NA
250	1079544	Y	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	10.3	8.8	80/18/2	367	12	NEG	0.93	18	0.7	138	3.4	0.78	6.4	3.6	98	38	45	8.9	NORMAL	NORMAL	NORMAL	1	NA
251	1084302	-	-	HMF ALTERED, B/L LOWER LIMB AREFLEXIA,B/L PLANTAR MUTE	NAD	B/L AE REDUCED IN LOWER ZONES	NAD	11.8	12.4	83/12/2	418	43	NEG	1.23	47	0.45	129	0.58	0.44	5.3	1.95	75	20	13	9.1	LACTIC ACIDOSIS	NORMAL	NORMAL	BLOOD C/S:STAPH AUREUS	NEG
252	1094189	-	-	HMF ALTERED,MENINGEALSIGNS PRESENT	NAD	NAD	NAD	12.3	7.8	87/13/10	223	45	NEG	1.25	22	0.4	129	4	0.3	7.8	4.5	88	32	65	9	NORMAL	NORMAL	NORMAL	1	NEG
253	1081599	-	-	HMF ALTERED	NAD	NAD	NAD	11	12.1	88/10/2	145	40	NA	1.13	40	0.5	133	4	0.67	7.1	3.9	160	67	56	9.1	NORMAL	NORMAL	NORMAL	1	NEG
254	1114293	-	-	HMF ALTERED,GENERALISED RIGIDITY	NAD	NAD	NAD	9	10.3	65/25/10	165	21	NEG	1.29	15	0.7	145	4.2	2.08	5.4	2.54	78	34	54	7.2	LACTIC ACIDOSIS	NORMAL	NORMAL	URINE C/S: KLEBSIELLA	NA
255	1107438	Y	-	HMF ALTRED,LEFT PTOSIS,RIGHT ULPOWER 4/5	NAD	NAD	NAD	12.4	8.7	66/22/7/4	321	27	NEG	1.07	12	0.4	135	3,7	0.24	7	3.9	78	78	90	7.8	NORMAL	NORMAL	NORMAL	1	NA
256	1138940	-	-	HMF ALTERED	NAD	NAD	NAD	12.2	11	68/20/12	176	56	NEG	1.23	12	0.2	138	4.3	0.13	5.9	2.62	88	32	34	9	NORMAL	NORMAL	NORMAL	1	NEG
257	1137033	Y	-	HMF ALTERED, B/LPLANTAR MUTE, B/L PUPILS NOT REACTING	NAD	NAD	NAD	13.4	18.2	88/10/2	327	38	NA	1.41	22	0.4	162	4	0.2	7.2	4.3	76	22	34	7.9	LACTIC ACIDOSIS	NORMAL	NORMAL	1	NA
258	1137166	-	-	HMF ALTERED,NECK RIGIDITY	NAD	NAD	NAD	10.5	12.3	86/12/2	167	45	NEG	1.34	28	0.68	122	3.4	0.87	5.6	3.5	183	57	65	8.7	NORMAL	NORMAL	NORMAL	1	NA
259	965022	-	-	HMF ALTERED	NAD	NAD	NAD	7.4	9.8	78/10/9	150	11	NEG	1.33	58	4.35	137	3.6	1.14	6.5	3.9	180	56	78	7.1	ACIDOSIS	NORMAL	NORMAL	URINE C/S:KLEBSIELLA	NA
260	726920	-	-	HMF ALTERED	NAD	NAD	NAD	10.4	7	34/60/10	189	34	NEG	1.67	91	1.43	139	4.77	0.03	7.4	3.69	94	24	12	9.4	NORMAL	NORMAL	NORMAL	1	NEG
261	1119623	-	-	HMF ALTERED	NAD	B/L RONCHI PRESENT	NAD	14.2	9.6	94/4/2	120	17	NEG	1.21	20	0.57	140	4.9	2.23	5.9	3.25	60	39	26	8.9	METABOLIC ALKALOSIS, NO TYPE 2 RRF	EMPHYSEMATOUS CHANGES/RIGHT LZ PATCH	ABNORMAL	ET C/S: KLEBSIELLA	NEG
262	1111684	-	-	HMF ALTERED,LEFT HEMIPARESIS,PROPTOSIS	NAD	NAD	NAD	10.2	10.9	58/26/10	174	45	NA	1.21	41	1.44	129	4.9	1.39	5.7	1.37	820	63	27	9	ACIDOSIS	NORMAL	NORMAL	1	NEG
263	1120428	-	-	HMF ALTERED	NAD	NAD	NAD	7.7	16	72/20/8	365	60	NEG	1.33	44	1.03	138	3.52	0.4	6.2	2.73	135	20	23	8.7	ALKALOSIS	PERI HILAR OPACITIES	NORMAL	WOUND SWAB: STAPHYLOCOCCUS	NA
264	1120986	-	-	HMF ALTERED, NECK RIGIDITY PRESENT	NAD	NAD	NAD	11	10.8	72/18/8/2	167	78	NEG	1.07	12	0.4	139	4	0.67	6.5	3.24	99	116	279	8	NORMAL	NORMAL	NORMAL	1	NA
265	1119436	-	-	HMF ALTERED	ABNORMAL	B/L CREPTS PRESENT	NAD	9.8	42	92/7/1	190	33	NEG	1.33	30	0.6	138	4	1.98	5.2	1.9	290	134	278	7.1	ACIDOSIS	B/LDIFFUSEINFILTERATES	ABNORMAL	BLOODC/S: ENTEROCOCCUS	NA
266	1060293	-	-	HMF ALTERED, MOVING ALL 4 LIMBS	ABNORMAL	B/L AIR ENTRY REDUCED	NAD	7.8	29	90/8/2	128	56	NEG	1.28	87	5.3	132	4.1	0.65	5.9	2.66	167	45	78	8.7	ACIDOSIS	NORMAL	NORMAL	1	NEG
267	1133070	-	-	HMF ALTERED,MENINGEAL SIGNS PRESENT	NAD	NAD	NAD	12.1	8.5	71/35/8	167	76	NA	1.32	20	0.8	137	3.8	0.54	6.9	3.47	88	22	32	8.3	NORMAL	NORMAL	NORMAL	1	NA
268	1128159	-	-	HMF ALTERED,B/L PLANTAR UPGOING	NAD	NAD	NAD	9	8	70/28/2	178	5	NA	1.25	34	0.5	136	3.5	0.4	6.7	3.7	126	54	45	8.6	NORMAL	NORMAL	NORMAL	1	NA
269	955938	-	-	HMF ALTERED	NAD	NAD	NAD	11.2	7.8	80/12/8	187	88	NA	1.23	27	0.3	135	4	0.2	6.8	3.4	78	46	56	8	NORMAL	NORMAL	NORMAL	1	NA
270	1133584	-	-	HMF ALTERED	NAD	SPASM	NAD	11.3	15.8	94/4/2	140	29	NA	1.19	32	1.35	129	4.4	1.25	5.3	3.09	66	83	40	6.7	ACIDOSIS	INFILTRATES	NORMAL	1	NEG
271	1126690	-	-	HMF ALTERED, B/LPLANTAR MUTE	NAD	NAD	NAD	9.7	15.9	77/11/10	249	11	NEG	1.19	15	0.64	145	3	0.33	5.5	3,01	70	59	59	7.1	NORMAL	NORMAL	NORMAL	1	NEG

S.NO	MRD NO	Leptospira	Dengue	Scrub Typhus	Other Investigations	CSF CYTOLOGY_TLC	NEUTROPHILS	LYMPHOCYTES	GLUCOSE	S. GLUCOSE	PROTEIN	CHLORIDE	GRAM STAIN	INDIA INK PREPARATION	BACTERIAL ANTIGEN	CULTURE SENSITIVITY	ADA	CBNAAT	HSV PCR	CT HEAD_FOCAL HYPODENSITIES	CT HEAD HYDROCEPHALUS	CT HEAD_DIFFUSE CEREBRAL OEDEMA	CT HEAD INFARCT	CT HEAD OTHERS	CT HEAD NORMAL	MRI LEPTOMENINGITIS	MRI VASCULITIC INFARCT
238	1034107	NA	NA	NA	-	2	0	0	68	145	48	118	-	-	-	-	1.32	-	-	N	N	N	N	N	N	Y	N
239	1042343	NA	NA	NA	-	20	90	10	53	90	70	123	-	-	-	-	8.94	-	NEGATIVE	N	N	N	N	N	Y	N	N
240	1043963	NA	NA	NA	-	20	5	95	42	90	306	119	-	-	-	-	46	-	-	N	N	N	N	ARACHNOID CYST	N	Y	Y
241	1045405	NA	NA	NA	PCT 65	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
242	1050401	NA	NA	NA	PCT 53	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
243	1059160	NA	NA	NA	-	2	0	0	56	90	88	120	-	-	-	-	11	-	-	N	N	N	N	N	N	N	N
244	1022929	NA	NA	NA	NCCT KUB:PYELITIS	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
245	1065734	POSITIVE	NEG	NEG	-	2	0	0	85	110	25	135	-	-	-	-	1.36	NEGATIVE	-	N	N	N	N	N	Y	N	N
246	891968	NEG	NEG	NEG	-	300	0	100	49	102	33	130	-	-	-	-	2.1	-	POSITIVE	N	N	N	N	N	N	N	N
247	1020516	NA	NA	NA	FUNSUS:PAPPILODEMA	60	5	95	32	89	160	108	-	-	-	-	16	-	-	N	Y	N	N	N	N	Y	N
248	1072015	NA	NA	NA	-	80	10	90	111	132	75	122	-	-	-	-	10	-	POSITIVE	N	N	N	N	N	N	N	N
249	993575	NA	NA	NA	-	50	30	70	22	182	127	101	-	-	-	-	28	-	-	N	N	N	N	N	N	Y	Y
250	1079544	NA	NA	NA	-	350	0	100	55	99	100.4	112	-	-	-	-	9.2	-	-	N	N	N	N	N	N	N	Y
251	1084302	NEG	NEG	NEG	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
252	1094189	NA	NA	NA	-	2	0	0	77	99	67	109	-	-	-	-	1.2	-	NEGATIVE	N	N	N	N	N	N	N	N
253	1081599	NA	NA	NA	-	70	70	30	78	110	45	131	-	-	-	-	2.56	-	-	N	N	N	N	N	N	N	N
254	1114293	NA	NA	NA	-	2	0	0	62	108	94	96	-	-	-	-	1.2	-	POSITIVE	N	N	N	N	N	N	N	N
255	1107438	NA	NA	NA	-	100	0	100	26	90	132	108	-	-	-	-	68	-	-	N	N	N	N	N	N	Y	N
256	1138940	NA	NA	NA	-	40	0	100	13	96	282	108	-	-	-	-	23	POSITIVE	-	N	N	N	N	N	N	Y	Y
257	1137033	NA	NA	NA	-	120	0	100	57	98	193	131	-	-	-	-	16	-	-	N	Y	N	N	N	N	Y	N
258	1137166	NA	NA	NA	-	20	10	90	34	102	88	112	-	POSITIVE	-	-	7.9	-	-	N	N	N	N	N	N	Y	Y
259	965022	NA	NA	NA	PCT 13	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
260	726920	NA	NA	NA	FUNGITELL 523/GALACTOMANNAN 1.21	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
261	1119623	NEG	NEG	NEG	BLOOD ALCOHOL LEVELS: NORMAL	2	0	0	93	119	88	120	-	-	-	-	9.75	NEGATIVE	-	N	N	N	N	N	Y	N	N
262	1111684	NA	NA	NA	-	-	-	-	-	-	-	-	-	POSITIVE	-	-	-	-	-	N	N	N	N	N	N	N	N
263	1120428	NA	NA	NA	PCT-0.95	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
264	1120986	NA	NA	NA	-	70	35	65	46	89	130	106	-	-	-	-	14	-	-	N	N	N	N	N	N	Y	N
265	1119436	NA	NA	NA	PCT >100,2D ECHO:INFECTIVE ENDOCARDITIS	8	90	10	89	102	54	110	-	-	-	-	2.3	NEGATIVE	NEGATIVE	N	N	N	N	N	Y	N	Y
266	1060293	NA	NA	NA	PCT 82	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	N	N	N
267	1133070	NA	NA	NA	-	50	0	100	19	96	259	119	-	-	-	-	17	-	-	N	N	N	N	N	N	Y	N
268	1128159	NA	NA	NA	-	2	0	0	54	90	134	107	-	-	-	KLEBSIELLA PNEUMONIAE	2.4	-	-	N	Y	N	N	N	N	Y	Y
269	955938	NA	NA	NA	-	50	30	70	35	88	128	108	-	-	-	-	39	-	-	N	N	N	N	N	N	Y	N
270	1133584	NA	NA	NA	PCT12	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N	N	N	N	N	Y	N	N
271	1126690	NEG	NEG	NEG	-	2	0	0	131	167	53	132	-	-	-	-	2.56	NEGATIVE	NEGATIVE	N	N	N	N	N	N	N	N

S.NO	MRD NO	MRI_RING ENHANCING LESIONS	MRI_MENINGIOENCEPHALITIS/ ENCEPHALITIS	MRI HYDROCEPHALUS	MRI_DIFFUSE CERBRAL OEDEMA	MRI NORMAL	MRI_OTHERS	EEG (if needed)	TUBERCULOUS MENINGITIS	2.VIRAL	3.DENGUE ENCEPHALOPATHY/ ENCEPHALITIS	4.PYOGENIC	5.MALARIA	6.SCRUB RELATED ENCEPHALOPATHY	7.SEPTIC ENCEPHALOPATHY	POST COVID ENCEPHALOPATHY	LEPTOSPIRA INDUCED ENCEPHALOPATHY	OTHERS	TREATMENT GIVEN	OUTCOME 1. RECOVERED, 2. DAMA,
238	1034107	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
239	1042343	N	N	N	N	N	-	-	N	N	N	Y	N	N	N	N	N	N	IV ANTIBIOTICS/STEROIDS/SUPPORTIVE	3
240	1043963	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
241	1045405	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/PERITONEAL DIALYSIS/VNTILATORY SUPPORT	3
242	1050401	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/DEBRIDEMENT/VENTILATORY SUPPORT/INOTROPES	3
243	1059160	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
244	1022929	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/IV FLUIDS	1
245	1065734	N	N	N	N	Y	-	-	N	N	N	N	N	N	N	N	Y	N	IV ANTIBIOTICS/SUPPORTIVE	1
246	891968	N	Y	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIBIOTICS/ANTIVIRALS	3
247	1020516	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/ANTI OEDEMA	1
248	1072015	N	Y	N	N	N	HERPES ENCEPHALITIS	-	N	Y	N	N	N	N	N	N	N	N	ANTIVIRAL	1
249	993575	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
250	1079544	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
251	1084302	N	N	N	N	Y	-	MODERATE DIFFUSE ENCEPHALOPATHY	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS,CLEXANE,IVIG	3
252	1094189	N	N	N	N	Y	-	-	N	Y	N	N	N	N	N	N	N	N	IV ACYCLOVIR/IV ANTIBIOTICS	1
253	1081599	N	Y	N	N	N	-	-	N	N	N	Y	N	N	N	N	N	N	IV ANTIBIOTICS/ATT/STEROIDS	1
254	1114293	N	N	N	N	N	TRANSIENT SPLENIAL LESION	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIVIRAL/ANTI EPILEPTICS/INOTROPES	1
255	1107438	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIS	1
256	1138940	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
257	1137033	N	N	Y	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/EVD DONE	3
258	1137166	N	N	N	N	N	-	-	N	N	N	N	N	N	N	N	N	CRYPTOCOCCAL MENINGITIS	LIPOSOMAL AMPHOTERICIN B/FLUCONAZOLE	2
259	965022	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
260	726920	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/ANTIFUNGALS/SUPPORTIVE	2
261	1119623	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS,DIURETICS,STEROIDS	1
262	1111684	N	N	N	N	N	BRAIN ABSCESS	-	N	N	N	N	N	N	N	N	N	CRYPTOCOCAL MENINGITIS	IV ANTIFUNGALS	3
263	1120428	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	3
264	1120986	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS/MANNITOL	1
265	1119436	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/HAART/SUPPORTIVE	1
266	1060293	N	N	N	N	Y	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/SUPPORTIVE	1
267	1133070	N	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
268	1128159	N	N	Y	N	N	-	-	N	N	N	Y	N	N	N	N	N	N	POLYMYXIN B/ZAVICEFTA	2
269	955938	Y	N	N	N	N	-	-	Y	N	N	N	N	N	N	N	N	N	ATT/STEROIDS	1
270	1133584	N	N	N	N	N	-	-	N	N	N	N	N	N	Y	N	N	N	IV ANTIBIOTICS/STEROIDS/SUPPORTIVE	1
271	1126690	N	N	N	N	N	-	-	N	Y	N	N	N	N	N	N	N	N	IV ANTIBIOTICS/ANTIVIRALS	1

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Date 20/1/2021

The Principal,
DMC,Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of **Dr. Mehak Arora**, a student of your college for **MD General Medicine** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Acute febrile encephalopathy – clinical profile and outcome

Supervisor
Dr. Rajesh Mahajan
Prof.,
Dept. of Medicine,
DMCH, Ludhiana.

Co-Supervisor
Dr. Veenu Gupta
Prof.,
Dept. of Microbiology,
DMCH, Ludhiana.

Dr. Rajinder K. Bansal
Prof.,
Dept. of Neurology,

The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

SP Singh

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. ***If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.***
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

ACUTE FEBRILE ENCEPHALOPATHY- CLINICAL PROFILE AND OUTCOME

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTOR OF MEDICINE

In

MEDICINE

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

NOVEMBER, 2019 DR. MEHAK ARORA

DEPARTMENT OF MEDICINE
DAYANAND MEDICAL COLLEGE AND HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	Acute Febrile Encephalopathy- Clinical profile and outcome
For the degree of	M.D. Medicine
Name of the candidate	Dr. Mehak Arora
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Introduction: Acute febrile encephalopathy is a clinical scenario where patient presents with history of a short febrile illness accompanied or followed by altered mental state. Encephalopathy is characterized by diffuse disease of brain that alters it's structure or function. CNS infections are the commonest cause of such presentations in hospitals after non traumatic coma. The etiological profile may vary amongst different geographical areas and seasons. Moreover, list of differentials in such patients is potentially long ranging from pyogenic meningitis, viral encephalitis, tubercular meningitis being the commonest. There are potentially treatable infectious processes that might be associated with significant morbidity and mortality, timely diagnosis and treatment are of utmost importance.

Aim of Study: To study etiological profile of patients presenting with fever accompanied or followed by altered mental status.

To study outcome of such patients with different etiologies for their presenting complaints.

Materials & methods: This will be a prospective study conducted on the patients presenting with acute febrile encephalopathy admitted in the Emergency Department of Dayanand Medical College and Hospital, Ludhiana, Punjab over a period of two years. Patients presenting with Acute Febrile Encephalopathy willing to participate in the study after an informed consent are to be included in the study.

Clinical Significance: Evaluate the various differential diagnosis in patients presenting with fever and altered sensorium and the effectiveness of the treatment modalities used reflected by the outcome in these patients.

Keywords: fever, altered mental status, febrile encephalopathy, encephalitis, CS Fanalysis, 2D MRI BRAIN, septic encephalopathy, pyogenic meningitis.

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Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"Acute Febrile Encephalopathy- Clinical profile and outcome"** exist in the Department of Medicine, Dayanand Medical College and Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate herself under our supervision and guidance.

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Place: _____

Date: _____

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
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Topic of Thesis	Acute Febrile Encephalopathy- Clinical profile and outcome
Likely date of appearing in PG examination	June 2022
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Approved: Yes / ~~No~~

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Approved: Yes / ~~No~~

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PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

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4	DATE OF ADMISSION OF COURSE	6thMAY,2019
5	TITLE OF THE TOPIC	Acute Febrile Encephalopathy – Clinical profile and outcome

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Acute febrile encephalopathy is a clinical spectrum where a short febrile illness is accompanied or followed by altered mental state and is characterized by diffuse disease of brain that alters it's structure or function manifested by a combination of coma, seizures, and/or decerebration.

In febrile illness, CNS infections are the commonest cause of such presentations in hospitals, etiological agent may be a bacterium, virus or a parasite. Significant seasonal and geographical variations have been found to be closely related to the etiological profile of the disease. Fever with altered mental state commonly results from bacterial meningitis, Japanese B encephalitis (JE), cerebral malaria (CM), and typhoid encephalopathy. In tropical countries like India, CM, JE, and bacterial meningitis are the common causes of AFE, while tuberculous meningitis (TBM) can present with subacute or chronic history.

In a similar background of febrile illness, encephalopathy can be precipitated by systemic infections or sepsis without cerebral inflammation i.e.in the setting of Sepsis Associated Encephalopathy where endogenous inflammation of the brain(encephalitis) is not the cause of fever and altered sensorium. Moreover, sepsis can lead to altered sensorium secondary to systemic complications like hypoglycemia, hypovolemia, hepatic or renal failure, hence these possibilities are to be kept as differentials before attempting to treat these patients.

It is important for the emergency clinicians to accurately diagnose and administer antimicrobials and adjunctive therapies to those who need them while attempting to wait for results of invasive testing. But at the same time irrational use of antibiotics should be avoided in patients who will not be benefitted by any such therapy (alternate etiology with similar presentation: for example severe rise in body temperature due to overproduction of heat, impaired dissipation or hypothalamic lesions due to non-infective CNS diseases).Conditions like Neuroleptic

Malignant syndrome though very rare should be kept in mind before reaching the diagnosis. Focal neurological deficits go in favor of Encephalitis(inflammation induced brain damage) as compared to other causes of encephalopathy but further investigations are required to confirm the diagnosis. Early decision making and rapid institution of therapy can be life-saving. In untreated cases, mortality is very high and the survivors are often left with disabling neurologic sequelae. Many a times even a detailed diagnostic workup may not identify a specific organism. Nevertheless a detailed examination and workup is warranted as many conditions, such as herpes simplex encephalitis (HSE) and cerebral malaria are eminently treatable. CT scan of the brain should be performed as a baseline investigation, to r/o contraindications of CSF and it may also be s/o meningeal enhancement (on contrast enhanced CT). Since meningeal enhancement is not a specific sign of meningitis and Acute Meningitis may present without showing abnormalities on CT, further investigations like CSF, MRI BRAIN to be considered afterwards. MRI in cases of HSV and JE may have characteristic findings. A GCS and/or a focal neurological deficit at hospital admission have been found to be independent clinical markers of bad outcomes among children with AFE. Raised intracranial pressure may be contributing to mortality in patients with meningoencephalitis, hence early diagnosis can be helpful. Most acutely ill febrile patients with encephalopathy can make complete neurological recovery once the underlying cause is identified and treated promptly and appropriately, but considerable skill and knowledge is required to distinguish the various groups. This study is being carried out to evaluate the patients presenting with AFE in a tertiary care center in northern India to understand the etiology, prevalence, any seasonal variation, and their outcomes and changes in etiology and outcome related trends as compared to the previous studies. This study is to be conducted over a period of 1 and half year.

6.2 REVIEW OF LITERATURE

Acute febrile encephalopathy is a clinical scenario where patient presents with history of a short febrile illness accompanied or followed by altered mental state. Encephalopathy is characterized by diffuse disease of brain that alters it's structure or function, list of differentials in such patients with fever and altered mental state is potentially long ranging from pyogenic meningitis, viral encephalitis, tubercular meningeal involvement to cerebral malaria, dengue viral encephalopathy, Leptospira related meningoencephalitis and aseptic meningitis, SAE (Sepsis Associated Encephalopathy, HIV related neuronal complications and other rare causes. A few studies conducted in same context are enlisted below.

In 1993, Durand M et al conducted prospective study on "Acute bacterial meningitis in adults. A review of 493 episodes." And found that major proportion of cases of acute bacterial meningitis in adults were nosocomial. Recurrent episodes of meningitis were frequent and overall mortality rate remained high.[1]

In 2001,Panagariya et al did a study on Herpes simplex encephalitis in North West India and found that Main clinical features were seizures (89%) and altered sensorium (100%). CSF examination was abnormal in 25 (90%) cases with (64%) patients. CT scan showed asymmetric frontotemporal lesions with or without haemorrhage in 18 (64%) cases. MRI was fairly characteristic with bilateral asymmetric frontotemporal lesions in all the 16 patients, in whom it was done. Out of the 28 patients, 18 reported within one week of the onset of symptoms. Of these, 13 patients had altered sensorium (grade I-II) and the disease was acute. Acyclovir was given to them immediately. 23% patients showed good recovery (Gr.-5), and 23% patients died. The remaining 5 patients in this group were stuporosed/comatosed on admission. They were given steroids along with acyclovir. Three patients (60%) survived with moderate residual deficits (Gr.-4) and two (40%) died. Out of 10 patients who reported after one week of onset of the first symptom, 3 were stuporosed/comatosed (grade III-IV) and they died early in the course of the disease, even after treatment with acyclovir and steroids.

Remaining 7 patients were treated with acyclovir alone. Four of them (57%) survived with moderate residual deficits (Gr.-4), while 3 (43%) patients died.[2]

In 2005, Bansal et al conducted a study on "To study the etiology and clinical profile of non-traumatic coma in children and to determine the clinical signs predictive of outcome." And concluded that etiology of coma in 60% cases was CNS infection (tubercular meningitis-19, encephalitis-18, bacterial meningitis-16, others-7); other causes were toxic-metabolic conditions (19%), status epilepticus (10%), intracranial bleed (7%), and miscellaneous (4%). 65 children survived, 11 were normal, 14 had mild disability, 21 had moderate disability and 14 were severely disabled and dependent. Survival was significantly better in patients with CNS infection (63%) as compared to those with toxic-metabolic causes (27%) and intracranial bleed.[3]

In 2007,Kumar et al conducted a study titled Dengue encephalopathy in children in Northern India: Clinical features and comparison with non dengue and reported that out of a total of 265 patients of AFE were enrolled over a 2 year period, HI test was positive in 15/49 (30.6%) and IgM in 52/238 (21.8%) patients thus tested. A total of 62 patients were positive for dengue antibodies by either test. Real time PCR assay for dengue virus genome was positive in 28/42 (69%) tested — 21/29 (72.4%) in CSF and 9/15 (60%) in serum. A total of 39 patients met the criteria for definite dengue infection of which only 2 fulfilled the WHO criteria for DHF. Comparing DE and non DE, rash, bleeding, swelling over body, and hepatomegaly were significantly more common and meningeal signs less frequent in DE. Mean platelet counts and serum albumen were lower and liver enzymes and INR were significantly higher in DE.[4]

In 2008, Karmakar et al conducted a study of A study of acute febrile encephalopathy with special reference to viral etiology on 151 patients with mean age of 3.21 ± 2.9 (range of mth-13 years) and male: female ratio of 1.71: 1. A diagnosis other than viral encephalitis was reached in 94 patients (62.3 %). Pyogenic meningitis was the most frequent diagnosis 51 (33.8 %) followed by tubercular meningitis 12 (7.9 %), and

cerebral malaria 8 (5.2 %) in the patient group of non-viral causes. Fifty-seven cases (37.3%) were suspected as viral encephalitis and mean age of the cases suspected as viral encephalitis was 2.8 ± 2.9 (Range 1 mth-10 yrs) with male: female ratio of 1.28: 1. Etiological diagnosis was reached or considered probable in 41 (72%) cases out of the suspected patients. The most common etiological agent identified was enterovirus 71 in 20 patients (35.1 %). The other viruses identified were mumps in 6 (10.5%), Japanese encephalitis in 5 (8.7%), and measles in 4 (7%) cases. MRI brain was done in 39 patients and was abnormal in 14 patients. Out of 57 cases of suspected viral encephalitis 10 patients expired within 48 hours, 2 > 48 hours and 19 patients had significant neurological sequels at discharge.[5]

In 2012, A Modi et al conducted a study on "The etiological diagnosis and outcome in patients of acute febrile encephalopathy: A prospective observational study at tertiary care center" and found that pyogenic meningitis was the leading cause of AFE, followed by AVE and cerebral malaria. The outcome in cases with AVE can be fatal or more disabling than other etiologies.[6]

In 2014, **Ashish Bhalla et al** conducted a study on "Acute febrile encephalopathy in adults from Northwest India and found that 70% had primary CNS infection as the etiology. A total of 33% patients had meningitis, 29.9% had evidence of meningoencephalitis, and 12.7% were diagnosed as sepsis-associated encephalopathy. These were followed by cerebral malaria, leptospirosis, and brain abscess as the cause of febrile encephalopathy in adults. Among the noninfectious causes, acute disseminated encephalomyelitis, cortical venous thrombosis, and neuroleptic malignant syndrome were documented in 2.36% each. In 11% of the patients, the final diagnosis could not be made in spite of the extensive investigations. His study demonstrated that acute febrile encephalopathy in adults is a heterogeneous syndrome with primary CNS infections being the commonest etiology.[7]

In 2015, Naderi Hamiz Raza conducted a study on "Investigating the Causes of Febrile Encephalopathy in Elderly Patients Admitted to Imam Reza Hospital in Mashhad during the 2013 to 2014" and found

that Infectious causes were responsible for febrile encephalopathy in 94 patients, of which 84% had an extra- cranial infection and neuro-infectious process was detected in 10%. The age of all 100 participants were 65 years and over consisting of 39 female and 61 male. Of them, only 34 patients underwent lumbar puncture, among them the cerebrospinal fluid (CSF) analysis was abnormal in 29.4%.[8]

In 2013, Anusha Deepthi et al did a study on acute febrile encephalopathy in children. In this study, AFE was higher among the age group of 6-14 years (54.7%) with most common cause being viral encephalitis (38%). Higher mortality rates were observed in CNS infections. Persistent seizures and hemiparesis were the most common causes of morbidity. GCS at initial presentation <7 was significantly associated with mortality, p value being 0.036.[9]

In 2017, Yerramilli A et al conducted study on the clinical outcomes and management of meningitis at a tertiary care centre. A total of 147 patients suspected to be having meningitis were included. The common types of meningitis in study were aseptic (39%), tuberculous (28%), pyogenic (28%), fungal (3%) and others (2%). The classical triad of headache with fever, neck stiffness, and altered mental sensorium was seen in 26% patients, and 83% had at least 2 out of these 4 symptoms. The incidence of seizures was found to be 63% in the presence of aseptic meningitis. Significant clinical improvement was seen in 89% of the patients suffering from aseptic meningitis.[10]

In 2006,ME Yeolekar, TH trivedi published an editorial on "Febrile Encephalopathy: challenges in Management" and concluded that clinician needs to consider a host of conditions of diverse origin, depending on geographic area, seasonal trend and recent spurts. Index of clinical suspicion indeed needs to be high. Initial empirical treatment after collection of blood/ CSF is justified. Treatment based on close continuous monitoring of clinical, hemodynamic and laboratory parameters with appropriate imaging can only help salvaging the patient and minimizing sequelae in the clinical challenge posed by acute febrile encephalopathy.[11]

The present study is being conducted to highlight the present scenario with regards to the etiological trends of acute febrile encephalopathy in a tertiary care centre in Northern India and assess the changing trends if present from the previous studies and assess the outcome of patients presenting with different etiologies with symptoms of fever and altered sensorium.[11]

In Novembor,2016, Avinash Aggarwal, Manish Gutch et al did a study named "State of Globe Acute Febrile Encephalopathy" and concluded that, patients can make complete recovery provided the underlying cause is treated timely. Efficiency and skill are required to correctly and timely diagnose the underlying etiology. A small but significant number of patients die, and some of the patients are left with neurologic sequelae. Delayed neurologic recovery and sequelae are also well recognized.[12] The fact that maximum mortality is seen in patients with AVE and SAE signifies that diffuse cerebral parenchymal involvement, lack of availability of specific drug for most of the viral infections, and multiorgan dysfunction in SAE possibly contribute to the overall increased mortality in these patients. Raised intracranial pressure in meningoencephalitis also contributes to mortality. Patients in whom a definitive diagnosis cannot be reached also show higher mortality

In 2015, Sukrirti Kumar et al did a study called Acute viral encephalitis clinical features and outcome: Experience from a tertiary center of North India and concluded that JE is the most common etiology identified followed by HSV encephalitis. The outcome in cases with AVE can be fatal or more disabling than other etiologies. Higher prevalence of JE is related to geographic and seasonal variations.[13]

In 2012, Nirdesh Jain et al did a case study on fatal combination of dengue shock syndrome with subdural hematoma. A 22-year-old male was admitted with high-grade fever since 1 week and headache since 3 days and unconsciousness since 1 day. History of head trauma was absent. Past history revealed that he had had dengue fever last year for which hospital admission was not required and he had recovered within a week. On examination, the patient was cyanosed with cool extremities and blood pressure was not recordable. On abdominal

examination, abdomen was distended; free fluid was present without having any organomegaly and bowel sounds were present. Neurological examination revealed no signs of meningeal irritations, a Glasgow Coma Scale (GCS) score of 4 (E1M2V1) and bilateral plantar reflex was non-elicitable. Computed tomography (CT) scan brain showed massive left parietal acute SDH obliterating left lateral ventricle with midline shift. Diagnosis of DSS was considered on clinical and investigational background. The patient was given fluid resuscitation and mechanical ventilation and, but the blood pressure was not picked up even after 3 hours of resuscitation. Subsequently, he was given vasopressor support. Next day, four units of platelets were transfused when his platelet count dropped to 52,000/mm 3. Despite all life-saving measures, he died.[14]

In 2010, J.M.K Murthy did a study on Neurological manifestations of Dengue and concluded that Encephalopathy is the most common neurological manifestation of dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS), and the pathophysiology is multi-factorial and includes cerebral edema, cerebral hemorrhage, hyponatremia, hepatic failure, renal failure and cerebral hypoxia Pathological studies in fatal cases of dengue showed nonspecific lesions, edema, vascular congestion, focal hemorrhages. In patients with dengue encephalopathy, magnetic resonance imaging (MRI) brain abnormalities included edema and scattered focal lesions, hemorrhage and cerebral edema.[15]

In 2002, PGE Kennedy and A. Chawdhary published an article on herpes simplex encephalitis and concluded that Both the mortality and morbidity of patients with HSE have been dramatically improved by the advent of the nucleoside analogue acyclovir (acycloguanosine) especially if it is initiated early, as soon as the diagnosis of HSE is suspected Acyclovir resistance in proven HSE has not been shown to occur in immunocompetent patients, although HSV resistance to acyclovir has been reported in the immunocompromised. Whether intravenous acyclovir treatment should be followed by the use of valacyclovir orally26 for an extended period is an issue that has yet to

be resolved. Despite these remarkable advances in treatment, HSE still has an unacceptably high mortality (20–30% with acyclovir) and morbidity. A better outcome is seen in younger patients below the age of 30 years and in those in whom the duration of encephalitis was four days or less and the Glasgow coma score was above 6 when acyclovir was started.[16]

In 2006, VM Kothari et al did a study on tropical infections in ICU and concluded that Fever with altered mental status is produced by bacterial meningitis, Japanese B encephalitis, cerebral malarial, typhoid encephalopathy and fulminant hepatic failure due to viral hepatitis. Subtle differences in features of the organ failure exist among these infections. The diagnosis in some of these diseases is made by demonstration of antibodies in serum, and these may be negative in the first week of the illness. Hence empiric therapy for more than one disorder may be justified in a small proportion of cases. In addition to specific anti-infective therapy, management of organ dysfunction includes use of mechanical ventilation, vasopressor drugs, continuous renal replacement therapy and blood products. Timely transfer of these patients to well-equipped ICUs with experience in managing these cases can considerably decrease mortality and morbidity.[17]

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To study etiological profile of patients presenting with fever accompanied or followed by altered mental status.
- To study outcome of such patients with different etiologies for their presenting complaints.

7. MATERIALS AND METHODS

This study will be conducted at Dayanand Medical College and hospital, Ludhiana. It will be an observational study, using data from patients admitted in DMCH, Medicine and Neurology wards presenting with acute febrile encephalopathy.

7.1 Source of Data

This will be an observational prospective study to be conducted on all the patients presenting with complaints of fever accompanied or followed by altered sensorium fitting into the inclusion criteria of the study in the Department of Medical Emergency of DMCH, Ludhiana over a period of 1 and half years from 1st Jan 2020 to 30th June 2021. Outcome of the patients will be assessed at the time of discharge.

7.2 METHOD OF COLLECTION OF DATA

All patients presenting with fever with altered sensorium either at onset or following fever and willing to participate in the study after giving informed consent will be included in the study. Appropriate history and clinical examination shall be done and Routine investigations like complete hemogram, RFT, LFT, RBS, Blood C/S, WIDAL and investigations which are a part of evaluation of the cause of such presentation like Tropical fever profile, CSF Analysis, Radio imaging will be done.

SAMPLE COLLECTION:

1. Blood Sample: Under all aseptic conditions, 10-20 ml of blood to be withdrawn from accessible veins and innoculated directly into culture bottles.
2. CSF sample will be collected aseptically by lumbar puncture into sterile glass vials and samples to be sent for Biochemical analysis, cytology and microbiological investigations.

   Direct examination will include wet mount preparation, India Ink preparation, Bacterial antigen and Cryptococcal antigen.

   Blood and body fluid specimens to be processed in BACTEC 9240 or BacT/Alert microbial detection system, incubation to be done for a maximum period of 7 days, smears will be prepared from positive blood culture bottles and Gram staining will be

done. The positive bottles will be subcultured on Blood Agar and MacConkey Agar plates at 37 degree celsius for 18-24 hours. Identification and AST (antibiotic sensitivity testing) of the isolates will be done by Vitek 2 System

3. For Tropical Fever serology, blood samples to be taken in vacutainers under all aseptic conditions and processing to be done as per standard kit protocol.

   The work up for Tropical fever will include:

   i. Dengue NS 1 Antigen
   ii. Dengue IgM
   iii. Scrub Typhus IgM
   iv. Leptospira IgM
   v. Malaria Antigen

INCLUSION CRITERIA

- age more than 18 years
- patients/attendants agreeing to participate in the study after signing informed consent
- patients presenting with fever of less than 15 days duration which is either accompanied or followed by altered mental status.(GCS<12)
- gender: either

EXCLUSION CRITERIA

- Consent not available
- age <18 years
- cerebral vascular accident followed by fever
- any metabolic cause which is independently explaining the presentation of altered sensorium.

DURATION OF STUDY: One and half year from 1st Jan 2020 to 30th June 2020.

STATISTICAL ANALYSIS

Data will be described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done

using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

NO

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Attached Herewith

8. LIST OF REFERENCES

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17. Kothari VM, Karnad DR, Bichile LS. Tropical infections in the ICU. J Assoc Physicians India. 2006;54:291-8.

PROFORMA

S.NO

MRD NO:

NAME:

AGE/SEX

S/O D/O:

RELIGION: H/M/S/C

OCCUPATION:

SOCIOECONOMIC STATUS:

ADDRESS:

URBAN/RURAL:

RESPONDENT: PATIENT/ATTENDANT

1. CHIEF COMPLAINTS:

a. FEVER:

b. ALTERED SENSORIUM:

c. HEADACHE:

d. VERTIGO:

e. BLURRING OF VISION:

f. ABNORMAL BODY MOVEMENTS:

g. WEAKNESS IN ANY PART OF BODY:

h. VOMITING:

i. OTHERS:

2. PAST MEDICAL ILLNESS HISTORY:

Signature of Candidate

Signature of Supervisor

3. FAMILY HISTORY:

4. PERSONAL HISTORY:

5. DRUG HISTORY:

6. GENERAL PHYSICAL EXAMINATION:

Blood Pressure:	Temperature:	
Pulse Rate:		
Respiratiry Rate:	Pallor:	GCS:
Clubbing:	Icterus:	Edema:
Petechiae/ Purpura:	Lymphadenopathy:	Cyanosis:

7. SYSTEMIC EXAMINATION:

a. CENTRAL NERVOUS SYSTEM:

Signature of Candidate **Signature of Supervisor**

b. CARDIOVASCULAR SYSTEM:

c. RESPIRATORY SYSTEM:

d. ABDOMINAL:

9. INVESTIGATIONS:

TEST	
Hb	
TLC	
DLC	
Platelets	
ESR	
PBF for Malaria	
PTI(INR)	
Urea/Creatinine	
S. Na/K	
S. Bilirubin	
Total Proteins	
S.albumin	
ALP	
SGOT/SGPT	
S. calcium	

Signature of Candidate **Signature of Supervisor**

9. OTHER INVESTIGATIONS:

a. ABG
b. Chest X-ray
c. ECG
d. Blood C/s and Urine C/s(if needed)
e. Serology
Malaria Antigen:
Leptospira:
Dengue :
Scrub Typhus:
f. others

g. CSF ANALYSIS

CSF CYTOLOGY	
CSF GLUCOSE/S. GLUCOSE PROTEIN: CHLORIDE:	
GRAM STAIN	
INDIA INK PREPARATION	
BACTERIAL ANTIGEN:	
CULTURE SENSITIVITY	
ADA/TBPCR(whenever possible)	

Signature of Candidate **Signature of Supervisor**

h. SPECIAL TESTS (if needed):

HSV PCR

JE PCR

CMV PCR

ENTEROVIRUS

RADIOLOGICAL INVESTIGATIONS:

CT HEAD

And/or

MRI BRAIN (with contrast if required)

j. EEG (if needed)

11. DIAGNOSIS:

12. TREATMENT GIVEN:

13: OUTCOME:

Signature of Candidate

Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....
.....am willing to participate in the study **"Acute Febrile Encephalopathy- Clinical profile and outcome"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

....
Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं_____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य "**Acute Febrile Encephalopathy- Clinical profile and outcome**" में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

"Acute Febrile Encephalopathy- Clinical profile and outcome" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ